COUNTWAY LIBRARY
HC 6DZR

BOSTON
MEDICAL LIBRARY
8 THE FENWAY

THE
ANATOMICAL RECORD

MANAGING EDITOR

EDWARD A. BOYDEN
University of Minnesota

ASSOCIATE EDITORS

CHARLES H. DANFORTH
Stanford University

WILLIAM U. GARDNER
Yale University

J. C. B. GRANT
University of Toronto

HAROLD L. WEATHERFORD
Harvard University

VOLUME 95

MAY, JUNE, JULY, AUGUST

1946

THE WISTAR INSTITUTE OF ANATOMY AND BIOLOGY
PHILADELPHIA, PA.

31563

BOSTON MEDICAL
FEB 1
LIBRARY

CATALOGUED
FEB 18 1947
F. K. M.

CONTENTS

No. 1 MAY, 1946

Margaret A. Kelsall. Anomalies in the small intestine and cecum of inbred strains of mice. One plate (twelve figures) 1

Sydney Sunderland. The innervation of the first dorsal interosseous muscle of the hand 7

J. Francis Hartmann, W. R. Fain and J. M. Wolfe. A cytological study of the anterior hypophysis of the dog with particular reference to the presence of a fourth cell type. One plate (fourteen figures) 11

Alden B. Dawson. The postpartum history of the corpus luteum of the cat. Three plates (twelve figures) 29

Robert E. Gregg. An arterial anomaly in the fetal pig. Four text figures and one plate (one figure) 53

Jane M. Oppenheimer. A case of atypical twinning in Fundulus heteroclitus. One figure 67

Book Reviews:

- L. J. Wells. Biological actions of sex hormones. By Harold Burrows .. 73
- R. R. Bensley. Physical chemistry of cells and tissues. By Rudolf Höber and others 75
- Wilson D. Wallis. An introduction to physical anthropology. By M. F. Ashley Montagu 77

No. 2 JUNE, 1946

Ernest Lachman. Is the concept of the ''anatomical position'' still tenable? A contribution to the subject of anatomical nomenclature 81

Margaret R. Lewis, Henry A. Sloviter and Philip P. Goland. In vivo staining and retardation of growth of sarcomata in mice 89

Martin Silberberg and Ruth Silberberg. Further investigations on the effect of the male sex hormone on endochondral ossification. Three plates (nine figures) 97

Captain Louis Emory. Method of duplicating anatomical specimens. Eight figures ... 119

J. Szepsenwol. A comparison of growth, differentiation, activity and action currents of heart and skeletal muscle in tissue culture. Three figures ... 125

William B. Atkinson and James H. Leathem. The day to day level of estrogen and progestin during lactation in the mouse. One text figure and one plate (six figures) ... 147

Victor M. Emmel. The intracellular distribution of alkaline phosphatase activity following various methods of histologic fixation. One text figure and one plate (seven figures) ... 159

P. P. H. De Bruyn. The amoeboid movement of the mammalian leukocyte in tissue culture. Two plates (twelve figures) ... 177

P. L. Johnson and G. Bevelander. Glycogen phosphatase in the developing hair. One plate (eight figures) ... 193

American Association of Anatomists. Proceedings of the fifty-ninth session, officers and list of members ... 201

No. 3 JULY, 1946

F. Gaynor Evans. The anatomy and function of the foreleg in salamander locomotion. Six figures ... 257

S. R. M. Reynolds. The relation of hydrostatic conditions in the uterus to the size and shape of the conceptus during pregnancy: A concept of uterine accommodation. Three figures ... 283

Anthony A. Pearson and Sarah Lea O'Neill. A silver-gelatin method for staining nerve fibers. One plate (six figures) ... 297

J. M. Essenberg. Glass and steel containers for human anatomy. Two figures 303

Bradley M. Patten. Fusion of notochord to neural tube in a human embryo of the sixth week. One plate (three figures) ... 307

D. H. Bishop and J. H. Leathem. Response of prepuberal male mice to equine gonadotropin ... 313

Irene A. Pogogeff and Margaret R. Murray. Form and behavior of adult mammalian skeletal muscle in vitro. Three plates (seventeen figures) ... 321

Edmond J. Farris. The time of ovulation in the monkey ... 337

Frank N. Low. An anomalous middle meningeal artery. One figure ... 347

S. L. Washburn. The sequence of epiphysial union in the opossum ... 353

No. 4 AUGUST, 1946

RAFAEL GRASSO. The action of thiourea on the intracellular colloid of the thyroid gland. Three plates (twelve figures) 365

EMIL MARO SCHLEICHER. On the "conical openings" in the wall of venous sinusoids and their relation to the so-called erythrogenic capillaries in the bone marrow of man. One plate (two figures) 379

PETER GRUENWALD. Embryonic and postnatal development of the adrenal cortex, particularly the zona glomerulosa and accessory nodules. Three plates (eighteen figures) 391

HADLEY KIRKMAN. A simian, deeply cleft, bilobed gall bladder with a "phrygian cap." Three text figures and two plates (thirty-four figures) 423

V. O. HURME. Old and "new" concepts on the structure of dentin (a bibliographic note) 449

M. A. SHAH AND MUBARIKA SHAH. The arterial supply of the vermiform appendix. One figure 457

AMERICAN SOCIETY OF ZOOLOGISTS. Proceedings, officers, constitution, by-laws and list of members, forty-second annual meeting, Saint Louis 461

ANOMALIES IN THE SMALL INTESTINE AND CECUM OF INBRED STRAINS OF MICE

MARGARET A. KELSALL[1]
Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

ONE PLATE (TWELVE FIGURES)

The incidence of visceral anomalies are seldom reported because of the difficulty in observing the internal organs of animals in large numbers as compared with the facility of noticing variations in external structures. For this reason, it was considered worthwhile to record variations in the small intestine and cecum of inbred strains of mice.

MECKEL'S DIVERTICULUM

Meckel's diverticulum, a persistent part of the yolk stalk (Bailey and Miller, '11), was the only gross anomaly observed in the small intestine. It occurred in ten of the 1,299 intestines examined, an incidence of 0.77%, which is slightly less than that reported for man (2–3%) (Lewis, '42). All mice included in this work were over 2 months old and were, therefore, well over the age at which the vitelline duct normally regresses in the mouse. There is some variation in the incidence of Meckel's diverticulum in the strains studied (table 1), but the numbers are not sufficient to justify establishing strain differences. Five diverticula were observed among the 739 males, an incidence of 0.67%; and five occurred in females, an incidence of 0.95%. The diverticula varied in length from a long process in one (fig. 1) to a knob-like structure in 5 (figs. 2, 4, 8, 9, 10). None of the diverticula were confluent

---

[1] Recipient of a Finney-Howell Medical Research Fellowship.

THE ANATOMICAL RECORD, VOL. 95, NO. 1
MAY, 1946

with the umbilicus or attached to the intestine by a fibrous band.

Relationships of the diverticula to the Peyer's patches were not consistent within a strain or between strains. The third Peyer's patch, as counted from the ileocecal junction, occurred at the base of the diverticulum in three intestines; and the fifth patch was at the base in two intestines. The remaining three diverticula on which relationships to Peyer's patches were recorded were located intermediately between two Peyer's patches.

TABLE 1

Incidence of Meckel's diverticulum in several inbred strains of mice.

STRAIN	NO. OF ANIMALS	MECKEL'S DIVERTICULA	%	FEMALES	MALES
C3H low	70	2	2.86	38	32
C3H high	202	3	1.49	55	147
C57 Black	288	3	1.04	122	166
Swiss	163	1	0.61	82	81
C	202	1	0.50	111	91
Dba	157	0	0.00	75	82
Dba 212	42	0	0.00	9	33
A	80	0	0.00	34	46
Total	1,299	10	0.77	560	739

A comparison of the three diverticula in the high tumor strain of C3H with the three in the C57 Black mice shows that there may be innate structural differences. The three diverticula in the C3H (figs. 5, 6, 7) were about the same length. They were a little longer than the three found in the C57 Black mice (figs. 8, 9, 10). In addition, a Peyer's patch was present at the base of each diverticulum in the C3H strain, whereas no patch occurred at the base of the diverticula in the C57 Black mice.

ANOMALOUS CECUM

An abnormally shaped cecum that could in no way be considered a functional variant of the normal cecum was found in an old female mouse belonging to strain C57 Black. The cecum in mice follows the general outline given in figure 11; variations in length and shape are marked and are produced by the amount of contained material and by the subsequent degree of contraction of the circular and longitudinal muscle layers. The cecum represented in figure 12 was the only anomaly found in the 1,299 ceca. The orifices of the ileum and colon entered at widely separated points instead of the normal proximal positions (fig. 11).

SUMMARY

Meckel's diverticulum and one abnormal cecum were the only gross anomalies found in the small intestine and cecum of 1,299 mice belonging to eight inbred strains. The incidence of Meckel's diverticulum was 0.77%.

LITERATURE CITED

BAILEY, FREDERICK R., AND ADAM M. MILLER 1911 Text-book of Embryology. William Wood and Co., New York.

LEWIS, WARREN H. (Editor) 1942 Anatomy of the human body by Henry Gray. 24th ed. Lea and Febiger, Philadelphia.

PLATE 1

EXPLANATION OF FIGURES

All drawings are enlarged approximately 1.5 times.

Figures 1 to 10 inclusive represent Meckel's diverticulum.

1 From a male mouse belonging to strain C. The location of the diverticulum in relation to Peyer's patches was not determined.

2 From a female mouse belonging to the Swiss strain. The diverticulum was located between the 6th and 7th Peyer's patches.

3 From a male mouse belonging to the low tumor strain of C3H. This anomaly was located between the 4th and 5th Peyer's patches.

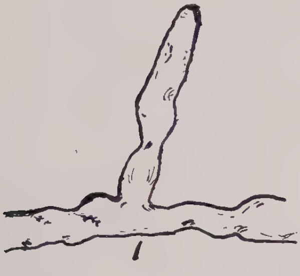
4 From a female mouse of the low tumor strain of C3H. The stipled area represents the 5th Peyer's patch located at the base of a diverticulum.

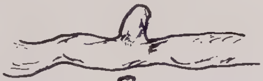
5, 6, and 7 Diverticula in strain C3H to show the uniform length and the presence of the 3rd Peyer's patch at the base in figures 5 and 6. The 5th patch is at the base of the diverticulum in figure 7.

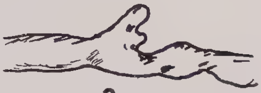
8, 9, and 10 Diverticula in strain C57 Black. The diverticulum in figure 8 was between the 3rd and 4th Peyer's patches.

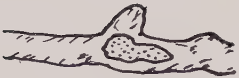
11 Normal cecum of a mouse. The lymphoid tissue is represented by the stipled area.

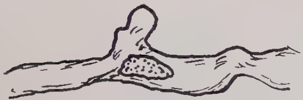
12 Anomalous cecum in a C57 Black mouse.

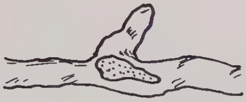

1

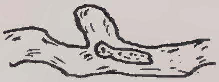

2

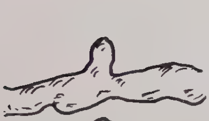

3

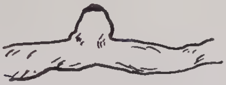

4

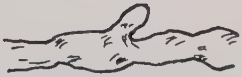

5

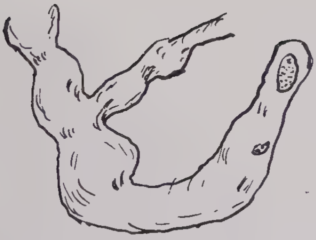

6

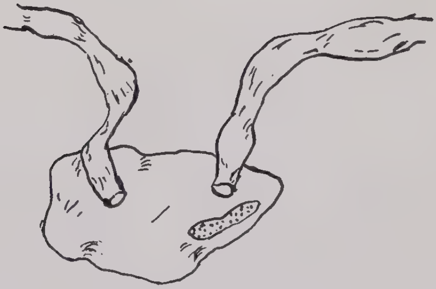

7


8


9


10


11


12

THE INNERVATION OF THE FIRST DORSAL INTEROSSEOUS MUSCLE OF THE HAND

SYDNEY SUNDERLAND

Department of Anatomy and Histology, University of Melbourne, Australia

It is generally accepted that the first dorsal interosseous muscle of the hand is innervated by the deep branch of the ulnar nerve (Macalister, 1889; Poirier and Charpy, '01; Piersol, '11; Morris, '15; Buchanan, '37; Wood Jones, '41; Gray, '42; and Cunningham, '43). McKenzie ('40), however, claimed that this muscle receives an accessory supply from the median nerve and in support of this advanced the following argument. Since, according to him, the interossei extend the terminal two phalanges but have no action on the metacarpo-phalangeal joints while the lumbricals flex the proximal phalanx but have no action on the interphalangeal joints then the ability to extend the interphalangeal joints of the index and middle fingers in ulnar nerve lesions could only be accounted for by the relevant interossei receiving an accessory supply from the median nerve. He, however, stated that he had no evidence of such a supply in "notes carried over a period of twelve years in the dissecting room." Curiously enough he made no reference to the condition of the first dorsal interosseous muscle in these lesions as revealed by palpation and electrical stimulation. In a previous communication ('45) the author demonstrated that the lumbricals and the extensor digitorum communis together can, in the absence of interosseous action, fully and effectively extend the terminal two phalanges and that this is the usual explanation of the preservation of interphalangeal extension of the index and middle fingers in ulnar nerve lesions. In the same paper McKenzie's original thesis, that the actions of the interossei and lumbricals are limited to the interphalangeal and metacarpo-phalangeal joints respectively, was examined in detail and disproved. Nevertheless, the author has, during an investigation of a series of peripheral nerve lesions, observed that this muscle is, on occasion, undoubtedly supplied from a source other than the ulnar nerve. The observations which form the subject of this paper concern the innervation of the first dorsal interosseous muscle by the median and superficial radial nerves.

OBSERVATIONS ON DISSECTED MATERIAL

The nerve supply to the muscle was traced by dissection in 100 dissecting-room specimens. In three of these the muscle was supplied exclusively, and in a fourth partially, by the median nerve. In a fifth specimen, an adult male Australian aborigine, the superficial radial nerve gave a terminal branch to the right first dorsal interosseous which also received an accessory supply from the deep branch of the ulnar nerve. It was not possible to determine whether the fibers from the superficial radial nerve were afferent or efferent. The supply from this nerve is of particular interest in view of the branch which the corresponding muscle in the foot frequently receives from the anterior tibial nerve. In the remaining 95 specimens the muscle was innervated solely by the ulnar nerve.

CLINICAL OBSERVATIONS

Patients with ulnar, median and radial nerve injuries occurring singly or in combination at and above the wrist are suitable for an investigation of this problem since in such cases each of the possible sources of supply to the muscle can, by a suitable selection of the cases, be separately isolated and studied.

In several patients with peripheral nerve injuries under the author's care at the 115th Australian Military Hospital and Repatriation Clinic, Melbourne, conditions were favorable for an investigation of the innervation of the first dorsal interosseous muscle. In each patient the injured nerve or nerves were either completely severed or there was indisputable clinical evidence that conduction had been completely interrupted for a long period prior to the onset of spontaneous recovery. The condition of the muscle observed in the nerve lesions severally set out was as follows:

	NUMBER OF LESIONS	STATE OF THE FIRST DORSAL INTEROSSEOUS ASCERTAINED BY PALPATION AND ELECTRICAL STIMULATION		
		Normal	Significant paresis	Paralysis
Ulnar nerve	41	3	2	36
Median nerve	17	15	2	0
Median and ulnar nerves	7	0	0	7
Radial nerve	39	39	0	0
Radial and ulnar nerves	3	0	0	3
Radial and median nerves	2	2	0	0

CONCLUSIONS

From these observations it is evident that on occasion the first dorsal interosseous muscle is partially or completely innervated by the median nerve. Furthermore, in one specimen fibers of an undetermined type were traced from the superficial radial nerve into the muscle through such an innervation appears to be rare. The preservation, in varying degree, of first dorsal interosseous muscle function in five patients with complete ulnar nerve lesions is proof only that, in these cases, this muscle was supplied either completely (3) or partially (2) from some other source. No attempt was made to isolate the nerve responsible for this supply by the procaine nerve block method (Highet, '42) but the accumulated evidence, anatomical and clinical, indicates that it was the median nerve. The anomalous innervation of this muscle should be kept in mind when estimating the state of conduction in injuries of the ulnar nerve on the basis of contraction of the muscle.

LITERATURE CITED

Buchanan 1937 Manual of Anatomy, 6th ed. London.

Cunningham, D. J. 1943 Text-Book of Anatomy, 8th Ed. London.

Gray, H. 1942 Anatomy, Descriptive and Applied. 28th Ed. London.

Highet, W. B. 1942 Procaine nerve block in the investigation of peripheral nerve injuries. J. Neur. and Psych., vol. 5, pp. 101–116.

MACALISTER, A. 1889 A Text-Book of Human Anatomy. London.

McKENZIE, W. C. 1940 The Action of Musles. 2nd Ed. London.

MORRIS, H. 1915 Treatise on Anatomy. 5th Ed. London.

PIERSOL, G. A. 1911 Human Anatomy. 3rd Ed. Philadelphia.

POIRIER, P., AND A. CHARPY 1901 Traité d'Anatomie Humaine. 2nd Ed. Vol. 2. Paris.

SUNDERLAND, S. 1945 The actions of the extensor digitorum communis, interosseous and lumbrical muscles. Am. J. Anat., vol. 77, pp. 189–217.

WOOD JONES, F. 1941 The Principles of Anatomy as Seen in the Hand. 2nd Ed. London.

A CYTOLOGICAL STUDY OF THE ANTERIOR HYPOPHYSIS OF THE DOG WITH PARTICULAR REFERENCE TO THE PRESENCE OF A FOURTH CELL TYPE [1]

J. FRANCIS HARTMANN,[2] W. R. FAIN AND J. M. WOLFE

Departments of Anatomy, Albany Medical College, Union University, Albany, New York and University of Minnesota, Minneapolis

ONE PLATE (FOURTEEN FIGURES)

The reported existence of a fourth cell type that is present in the anterior hypophysis of many animals in addition to the traditional acidophiles, basophiles and chromophobes is one of the more interesting aspects of hypophyseal cytology. In a study of cyclic histological variations in the anterior hypophysis of the female dog (Wolfe, Cleveland and Campbell, '33), such a fourth cell type was described. Cells of this type contained granules that stained selectively with erythrosin in the erythrosin-orange G-aniline blue method of Cleveland and Wolfe ('32). The erythrosin-stained cells were referred to as Type II, and were considered to be of a basophilic character.

Subsequently, Dawson and Friedgood ('38) discovered in the anterior hypophyses of female rabbits and cats an additional cell type with a special affinity for azocarmine. Their findings were followed by investigations revealing the presence of similar cells in the anterior lobes of several other species. They have been reported in the hypophyses of the opossum, ferret and monkey by Dawson ('38 and '42), in the armadillo by Oldham ('38), in various orders of birds by

---

[1] This study was aided by a grant from the Winthrop Research Fund Committee.

[2] Now at the University of Minnesota.

Rahn ('39) and Rahn and Painter ('41), in the newt by Copeland ('43) and in the garter snake by Hartmann ('44). The cells staining with azocarmine, however, were generally considered to be a second type of acidophile.

Meanwhile, the presence of an additional cell type in the anterior hypophysis of the dog has not been generally recognized. Because of this fact, and because the additional cell type of other species has been regarded as an acidophilic cell rather than basophilic, a careful restudy of the additional cell type in the anterior lobe of the dog was undertaken. The object was to determine whether or not the previously reported existence of a fourth cell type could be confirmed, and if so, to study the relation of this cell type to the usual acidophiles and basophiles. Two different fixatives and a variety of staining procedures were used in this analysis.

On the basis of the observations to be presented here, it has been concluded that a fourth cell type, granular in nature, does occur as a constant feature in the anterior hypophyses of both male and female dogs. A clear tinctorial separation of this cell type is relatively easy to accomplish, and the absence of intergrading forms makes it impossible to regard it as merely a variant of either the usual acidophilic or basophilic cell. In the opinion of the authors, however, its staining reactions under the experimentally varied technical conditions used in this study do not at present justify its classification unreservedly as either an acidophile or a basophile.

Dawson ('39) in a discussion of the possible existence of an additional chromophilic type, has pointed out that difficulties of classification arise, and the inadequacy of a system of classification based upon acid and basic staining reactions is well recognized by all workers in the field of hypophyseal cytology. It is not the purpose of this paper, however, to suggest a revision of terminology, but since the granules of the fourth cell type under discussion are stained selectively with erythrosin in the technique of Cleveland and Wolfe, it will be referred to provisionally as the erythrosin cell. Cells staining selectively with orange G will be referred to as acidophiles, while cells in which a blue color predominates with the techniques used will be designated as basophiles.

MATERIALS AND METHODS

The anterior hypophyses of 37 dogs, most of them males, were used for this study. Regaud's fluid and the sublimate-saline-formol solution recommended by Dawson and Friedgood ('38) were employed as fixatives. Some of the glands were fixed whole, but the majority of them were divided either into sagittal halves or into smaller pieces before fixation. They were dehydrated by the procedures outlined by Cleveland and Wolfe ('32) or by Dawson and Friedgood ('38), depending upon the fixation, and embedded in 60–62° Tissuemat (Fisher Scientific Co.). Serial sections were cut in either the horizontal or sagittal plane at 3 microns, one section being mounted per slide.

Three principal staining procedures, with certain modifications, were followed. These were (1) the azocarmine technique of Dawson and Friedgood ('38), (2) the erythrosin-orange G-aniline blue method of Cleveland and Wolfe ('32) and (3) the basic fuchsin mitochondrial technique of Fain and Wolfe ('44) which differentiates between mitochondria and the specific granules of the anterior lobe cells. The azocarmine technique was readily adapted to the hypophysis of the dog by increasing the staining time in azocarmine to 2½ hours at 56°C., and by the substitution of 1.0% aniline in 95% alcohol for the 0.1% aniline originally recommended for the first differentiation. This staining method was used only on material fixed in sublimate-saline-formol, all attempts to apply it to Regaud-fixed material having been unsuccessful. The method of Cleveland and Wolfe was modified by using 3.0% instead of 0.3% erythrosin, or by the use of either Goodpasture's polychrome methylene blue or the aniline blue suggested by Fain and Wolfe ('44) in place of the 1.0% aqueous aniline blue formerly used. By varying the staining times, good differentiation of cellular elements was obtained in all cases. Although on a few occasions 0.5% acid fuchsin was successfully substituted for erythrosin in this technique, for convenience the general procedure will be referred to as the erythrosin method. It was used on material fixed either in Regaud's fluid or in sublimate-saline-formol; in the latter case, chromation of the sections on the slides was omitted. The mitochondrial technique was used without modification, but only on material fixed in Regaud's fluid.

The study of individual cells in adjoining serial sections stained by different technical methods provided much useful information. Two microscopes were employed for the purpose, each equipped with achromatic condenser, apochromatic objectives, and compensating oculars. The Köhler system of illumination was used throughout. Successive sections from a given block of tissue, mounted on separate slides and stained by any two techniques, were studied and directly compared. A section through a cell stained by one method could then be matched with the adjacent section through the same cell stained by another method. It was frequently found that a particular cell could be identified in as many as four or five successive sections, and the cell-for-cell matching of the sections was therefore relatively easy and thoroughly reliable.

Three kinds of direct comparisons were made by the foregoing plan. (1) Sections through cells stained by the azocarmine method were compared with sections through the same cells stained by the erythrosin method after sublimate-saline-formol fixation. (2) Sections of cells stained by the mitochondrial technique were compared with those stained by the erythrosin method after Regaud fixation. (3) The individual stains of the erythrosin method were applied separately or in various combinations, and sections of cells so treated were compared with sections of the same cells in a fully-stained Cleveland and Wolfe preparation.

OBSERVATIONS AND CONCLUSIONS

The cytology of the additional cell type as it appears in sections of the dog hypophysis stained with the erythrosin method serves as a logical point of departure in attempts to analyze this cell in relation to the other chromophilic types. In a typical section subjected to any of the modifications of the erythrosin method already mentioned after Regaud fixation, the granules of the erythrosin cells (fig. 1) stain a bright red color that contrasts sharply with the lemon-yellow or orange-yellow color of the granules in the acidophiles (fig. 3). No intergrading forms between the acidophiles and the erythrosin cells have ever been observed. The erythrosin cells vary considerably in size and in density of granulation, ranging from cells as small as the smallest acidophiles to those as large as basophiles. The granules of these cells are of uniform size throughout, and are usually more closely packed in the smaller cells than in the larger ones. Occasional small cells with sparse granulation and occasional large cells with dense granulation have, however, been observed. When the erythrosin technique is used, the tinctorial reaction of the individual granules in these cells appears to be independent of the degree of their dispersion within the cell. Differences in density of granulation are reflected in differences in color density, but the tone of color is the same in all erythrosin cells (figs. 1–2).

The differentiation between the erythrosin cells and the acidophiles appears to be based on a genuine affinity of the granules of the former for erythrosin, rather than to artificial conditions of technique. This is shown by the fact that clear differentiation between the two types is obtained even when staining times are varied within wide limits. Thus if the staining time in erythrosin is increased several hundred per cent while keeping the time in orange G constant, the granules of the erythrosin cells are stained a bright red color, while those of the acidophiles take up only enough erythrosin to make them appear orange instead of lemon-yellow in color. Conversely, if the amount of exposure to orange G is increased, the granules of the acidophiles stain bright yellow while those of the erythrosin cells appear red with a tint of orange. In either case, however, the two cell types are clearly differentiated with no gradations between them.

It is well known that fixation in Regaud's fluid preserves the mitochondria, and attention was therefore directed to the possibility that the red granules in the erythrosin cells might be mitochondria rather than specific secretory granules. Several lines of evidence, however, argue against such a possibility. First, a large number of sections from several animals were stained by the basic fuchsin mitochondrial technique and carefully studied. If the red granules of the fourth cell type appearing in sections stained by the erythrosin method were mitochondria rather than specific granules, many cells completely filled with mitochondria should have been observed when the specific mitochondrial technique was used. However, this was found not to be the case.

The most convincing proof that the red-staining granules in the erythrosin cells are not merely mitochondria was obtained by making direct comparisons of individual erythrosin cells in alternate sections stained by the erythrosin method and the mitochondrial technique, respectively. It perhaps should again be emphasized that the identification of erythrosin cells in sections stained by the mitochondrial technique could be checked by observing the succeeding section of the same cell on an adjoining slide stained by the erythrosin method.

In sections stained by the mitochondrial technique, the staining reaction of the erythrosin cells varied rather widely. In some glands, the erythrosin cells in which the granules are closely packed stain a bluish pink color (fig. 5), while in more sparsely granulated cells the granules stain a dull blue (fig. 6) or even a bluish-gray color. Between these two extremes there are progressive intermediate gradations. In other glands, even cells in which the granules were closely packed stained a dull gray, which tended to vary to a bluish-gray in more sparsely granulated cells. The granules of the acidophiles are a uniform pink color with this technique (fig. 7), and those of the basophiles (fig. 8) are varying shades of blue. In all cells, however, the mitochondria are stained a brilliant fuchsin color, thus contrasting sharply with the specific granules of all cell types (figs. 5–8). Sections through individual erythrosin cells on one slide were matched with the neighboring sections through the same cells on the other slide. In no case was any correspondence found between the number and distribution of the erythrosin-stained granules of a given cell in one section and the number and distribution of mitochondria in the adjacent section through the same cell (figs. 1–2, 5–6).

Finally, there remains the fact that with the erythrosin technique, the mitochondria in the basophiles and chromophobes stain a pale orange color, which accounts for their being indistinguishable in the acidophiles and in the erythrosin cells. This circumstance is inconclusive in itself, but seems significant when regarded in conjunction with the foregoing observations, because it seems most unlikely that the mitochondria would stain red in the erythrosin cells while staining a pale orange color in the basophiles, chromophobes and, presumably, in the acidophiles. It was therefore concluded from these three lines of evidence that the erythrosin cells comprise a distinct granular cell type, and do not represent merely cells rich in mitochondria.

As a further step in the analysis of the erythrosin cell, it was desirable to determine whether or not the azocarmine technique of Dawson and Friedgood ('38) would demonstrate the presence of cells staining selectively with azocarmine in the anterior hypophysis of the dog, and if so, whether or not the cells staining selectively with azocarmine would prove to be identical with the erythrosin cells. As already stated, adaptation of the azocarmine technique to the anterior lobe of the dog was not difficult. In sections stained by this method, cells stained with azocarmine were present (fig. 10) and sharply distinguished from the orange cells (fig. 11), basophiles (fig. 12) and chromophobes. It was further found that the erythrosin method, when used after sublimate-saline-formol fixation, differentiated the cellular elements of the anterior lobe fully as well as when Regaud fixation was used. These conditions made possible a cell-for-cell comparison of the results of the azocarmine and the erythrosin techniques. A large number of cells were compared, and in every case a cell found to be selectively stained with azocarmine in one section was stained selectively with erythrosin in the adjacent section (figs. 9–10). The granules of the acidophiles and basophiles were stained orange and blue respectively with both techniques. With either staining method, the fourth cell type was tinctorially distinct, and not linked to the other chromophilic types by any cells of intermediate color.

These findings immediately bring up the question of the relationship between carmine cells and erythrosin cells. Apparently in the anterior lobe of the dog erythrosin cells and carmine cells are identical, but this does not mean that the erythrosin cells of the dog hypophysis are necessarily identical with the carmine cells of other species.

A possible approach to the problem of whether the erythrosin cells are acidophilic or basophilic in nature is provided by omitting any one or two individual components of the erythrosin technique and comparing sections so treated with adjacent sections fully stained. Individual cells can be identified in the fully stained section and matched against successive sections through the same cells treated with one stain only, or with any two of the stains used in the erythrosin method; thus it is possible to study the response of all cells to the separate stains.

If erythrosin alone is used, the erythrosin cells are stained a bright red, the acidophiles are a very pale yellowish color (perhaps imparted to them by the chromation process before staining), while the basophiles are pink to red, some of them being nearly as red as erythrosin cells. These findings have been repeatedly verified by cell-for-cell matching with adjacent fully stained sections, thus making positive identification of the individual cells involved. The fact that the basophiles have a greater affinity for erythrosin than do the acidophiles has a possible bearing on the classification of the erythrosin cells, to be discussed in a subsequent section.

If orange G alone is used, all tissue elements are stained yellow in varying degrees. The erythrosin cells stain with the greatest intensity, with acidophiles and basophiles taking up approximately equal amounts of the dye. If the sections are stained successively in orange G and aniline blue, however, the situation is quite different. The acidophiles are stained a bright lemon-yellow color, the basophiles are pale to deep blue, while the erythrosin cells are now a dull yellow with a tinge of blue (fig. 14). It could not be determined with certainty whether the blue dye taken up by the erythrosin cells had actually been retained by the granules or by the ground cytoplasm of these cells.

In sections stained only with erythrosin and aniline blue, the erythrosin cells are stained deep red, while the acidophiles are pale pink with a tinge of orange. Differentiation between these two types was never difficult, and identification of individual cells was always verified by direct comparison with the adjacent, fully stained section. The basophiles in these preparations were stained blue.

DISCUSSION

The constant appearance of an additional tinctorial cell type in the anterior hypophysis of the dog under controlled technical conditions leaves little room to question it as a distinct cytological entity. Cells of this group are characterized by unique staining affinities that make it possible to differentiate them from other chromophiles with a complete absence of intergrading forms. This circumstance compels the conclusion that the erythrosin cell in the anterior hypophysis of the dog cannot be viewed as a mere variant of another chromophilic type. It is unnecessary to emphasize the fact that failure to avoid technical hazards can result in the introduction of errors in a study of this kind. Hence, throughout the course of the work, every precaution was taken to exclude faulty or artificial conditions of technique, and it was felt that the appearances observed were reliable. It has been justly argued that too much should not be concluded from staining reactions alone, but they nevertheless constitute the most useful approach to many problems of microscopic anatomy, and they frequently serve as the only available criteria for certain cellular details.

In discussing the possible relationship of the erythrosin cell to the other cells of the anterior lobe of the dog, there is a natural tendency to assume that it is either an acidophilic or a basophilic type, since the chromophilic cells of the anterior hypophysis have traditionally been classified in one or the other of these categories. At the outset, however, it might be stated that the terms ''acidophilic'' and ''basophilic,'' inasfar as they pertain to the cells of the anterior lobe, have become almost meaningless, since many anterior lobe stains use only acid dyes. Indeed, in the recently published mitochondrial technique of Fain and Wolfe ('44), the acidophilic granules and the mitochondria are stained by basic fuchsin while the basophilic granules are colored by an acid dye, aniline blue. This difficulty in terminology has been recognized previously by Bailey ('28) who suggested that the terms alpha and beta cells, respectively, be used instead of the usual acidophiles and basophiles.

In the present paper the designation of the additional cell type has been based on its reaction to the particular technique used in its demonstration. Therefore, since this cell is selectively stained by erythrosin, it has been provisionally referred to as the erythrosin cell, although it could with equal justification be designated as a carmine cell or as an acid fuchsin cell. Such a makeshift nomenclature is of course undesirable in the long run, but until the diverse staining possibilities of the anterior lobe are more fully explored, it seems impossible to suggest a suitable terminology.

In spite of the inadequacy of our present terminology, it is interesting to compare the erythrosin cell with the acidophile and basophile and attempt to determine whether it should properly be classified as either an additional acidophilic or basophilic type. As previously mentioned, this cell can be selectively stained by any one of three acid stains. Certain additional information concerning its staining capacities may also be obtained by omitting various steps in the erythrosin method. When erythrosin alone is used, the fourth cell type is more intensely red than either of the other chromophilic types, and when the section is stained only with orange G, the additional cell is a brighter yellow than any of the other cells, even including the acidophiles.

Probably more definite information was gained from sections stained with orange G and aniline blue than in any other way. In this case, the two particular stains that are chiefly concerned in the final coloration of the acidophiles and basophiles in the erythrosin method were being dealt with. It was felt that under these conditions the selective affinity, if any, of the erythrosin cell for either of these stains could be determined. Unfortunately, the staining reaction of the erythrosin cells in such sections was not clear-cut. They stained a dull yellow color, but many cells also showed a tinge of blue. Although it seemed quite definite that a certain amount of orange G had been retained by the granules of the erythrosin cells, it could not be ascertained with any degree of satisfaction whether the slight bluish coloration of the cells was due to aniline blue retained by the actual granules or by the ground cytoplasm. It did seem clear, however, that these cells had more affinity for orange G than for aniline blue. These facts, therefore, taken alone, would probably justify the classification of the erythrosin cell as an acidophilic type.

On the other hand, there is some evidence that may be interpreted as showing a certain relationship or similarity between the erythrosin cells and the basophiles. At the outset, it should be stated that the present observations on the basophiles of the anterior lobe of the dog are incomplete and that further study is necessary if a fuller understanding of the cytological nature of these cells is to be obtained. In sections stained by the full erythrosin method, it was observed that the cytoplasm of the basophiles often contained reddish material. In some basophiles, this material appeared structureless in nature and was observed to occur in the form of large irregular masses or aggregations. Such cells also usually contained distinct blue granules (fig. 4). The masses or aggregations of reddish substance varied in size in different basophiles and sometimes occupied half the sectional area of the cell, with the granules being disposed elsewhere in the cytoplasm. Small numbers of basophiles have been observed to contain this erythrosin-stained material but no blue granules, and there were usually some basophiles which contained none of the reddish material (fig. 13). In cells which were altogether lacking in blue granules, the red substance was often widely dispersed in the cytoplasm and usually occurred in the form of coarse irregular particles which showed considerable tendency to fuse, although in occasional cells it appeared granular in nature. The simplest explanation for the occurrence of reddish material in the basophiles would be that clumps of mitochondria were staining with erythrosin. However, a careful matching of single cells in alternate sections stained by the erythrosin method and by the mitochondrial technique demonstrates that the reddish material in the basophiles stained by the erythrosin method are not masses of mitochondria. In the mitochondrial preparations it was found that these aggregations of reddish material were stained the same color as the rest of the cytoplasm. Although mitochondria were observed to occur in or near these masses, they were almost invariably less abundant in this region of the cell than elsewhere (figs. 4 and 8). Added to this is the fact that in sections stained by the erythrosin method, the mitochondria, although not prominent, are never stained red but dull orange. These findings, therefore, indicate that mitochondria are not responsible for the reddish material found in basophiles stained by the erythrosin method. The nature of this material is not known, but the fact that in some basophiles a cytologic constituent other than mitochondria shows an affinity for erythrosin, makes it impossible to rule out a certain similarity between the erythrosin cells and the basophiles. This fact, combined with the finding that in sections stained with orange G and aniline blue, some cytoplasmic constituent of the erythrosin cells may take up slight amounts of aniline blue, has prevented the making of any definite classification of this fourth cell type at the present time.

The appearance of the erythrosin cell in sections stained by the mitochondrial technique should be considered more fully. It may be stated that the specific granules of these cells were not satisfactorily stained by this method, and if this technique alone had been used, or if sections through individual cells had not been checked against adjoining sections of the same cells stained by the erythrosin method, it is possible that many of the erythrosin cells would not have been recognized. For instance, the erythrosin cells which were well packed with granules stained a dull brick-red, or often a bluish-pink (fig. 5). Erythrosin cells in which the granules were more widely dispersed took a fainter stain and here the granules appeared to stain a dull blue color (fig. 6). Often the granular nature of such cells might have been overlooked unless the cells had been checked against adjoining sections stained by the erythrosin method. It can therefore be seen that when the mitochondrial technique was used, the granules of the erythrosin cells were variable in staining reaction, inasmuch as this cell showed no clear-cut affinity for either basic fuchsin or aniline blue, but apparently had the capacity to take up small amounts of both. In contrast to the variability in the staining reaction of other constituents of the cytoplasm, the mitochondria invariably stained a brilliant fuchsin color in these cells (figs. 5–6). In our opinion, most of the erythrosin cells can be identified when studied by this method alone, but it should be pointed out that certain cells might prove confusing and that mistakes in identification, particularly in the sparsely granulated cells, might be made. Therefore, the basic fuchsin mitochondrial technique when used alone is considered unsatisfactory for the study of the anterior hypophysis of the dog; used in conjunction with the erythrosin method or a similar technique it is considered extremely valuable since it differentiates between mitochondria and specific granules, and thus demonstrates that the red-staining granules in the erythrosin cells are true secretory granules.

The fact that the erythrosin cells stain so poorly with the mitochondrial technique, while both the acidophiles and basophiles stain quite brilliantly, is considered further evidence that the erythrosin cell is dissimilar to both acidophiles and basophiles and that a definite classification should be postponed.

It is perhaps unnecessary to add that the conclusions reached in this study are felt to apply only to the cells in the anterior hypophysis of the dog. Whether or not they would apply to the anterior lobe cells of other species is a matter outside the scope of the present paper.

SUMMARY

1. A careful restudy of the anterior hypophysis of the dog has been made with special reference to a previously reported fourth cell type, provisionally designated as the erythrosin cell.
2. Two different fixatives and a variety of staining methods were employed, and all observations were checked by a serial study of individual cells in successive sections stained by different techniques.
3. The presence of the erythrosin cell, as a fourth cell type, granular in nature, and appearing as a constant feature in the anterior hypophysis of the dog, has been confirmed.
4. The clear tinctorial differentiation of this cell type is relatively easy to accomplish, and the absence of intergrading forms makes it impossible to regard it as a variant of either the usual acidophile or basophile.
5. Direct comparison of cells in sections stained specifically for mitochondria with adjacent sections through the same cells stained by the erythrosin method proves that the granules of the additional cell type are specific granules rather than mitochondria.
6. The erythrosin cells are identical with cells staining specifically with azocarmine in the anterior hypophysis of the dog.

LITERATURE CITED

BAILEY, P. 1928 The structure of the hypophysis cerebri of man and of the common laboratory mammals. Special Cytology, 1st edition, Paul B. Hoeber, Inc., New York, Section 15, pp. 487–499.

CLEVELAND, R., AND J. M. WOLFE 1932 A differential stain for the anterior lobe of the hypophysis. Anat. Rec., vol. 51, pp. 409–413.

COPELAND, D. E. 1943 Cytology of the pituitary gland in developing and adult Triturus viridescens. J. Morph., vol. 72, pp. 379–409.

DAWSON, A. B. 1938 The epithelial components of the pituitary gland of the opossum. Anat. Rec., vol. 72, pp. 181–193.

——— 1939 Differential staining of the anterior pituitary gland of the cat. Stain Tech., vol. 14, pp. 133–138.

——— 1942 Some morphological aspects of the secretory process. Fed. Proc., vol. 1, pp. 233–240.

DAWSON, A. B., AND H. B. FRIEDGOOD 1938 Differentiation of two classes of acidophiles in the anterior pituitary of the female rabbit and cat. Stain Tech., vol. 13, pp. 17–21.

FAIN, W. R., AND J. M. WOLFE 1944 A cytological stain for the anterior pituitary gland involving the use of basic fuchsin. Anat. Rec., vol. 90, pp. 311–314.

HARTMANN, J. F. 1944 Seasonal cytological changes in the anterior hypophysis of the garter snake. Am. J. Anat., vol. 75, pp. 121–149.

OLDHAM, F. K. 1938 The pharmacology and anatomy of the hypophysis of the armadillo. Anat. Rec., vol. 72, pp. 265–291.

RAHN, H. 1939 The development of the chick pituitary with special reference to the cellular differentiation of the pars buccalis. J. Morph., vol. 64, pp. 483–517.

RAHN, H., AND B. T. PAINTER 1941 A comparative histology of the bird pituitary. Anat. Rec., vol. 79, pp. 297–312.

WOLFE, J. M., R. CLEVELAND AND M. CAMPBELL 1933 Cyclic histological variations in the anterior hypophysis of the dog. Zeit. Zellforsch., vol. 17, pp. 420–452.

PLATE 1

EXPLANATION OF FIGURES

Drawings by Miss Virginia E. Moore, Medical Art Shop, University of Minnesota

All figures × 1350

1 Densely granulated erythrosin cell. Regaud fixation, Cleveland and Wolfe stain.

2 Sparsely granulated erythrosin cell. Regaud fixation, Cleveland and Wolfe stain.

3 Typical acidophile. Regaud fixation, Cleveland and Wolfe stain.

4 Basophile with reddish substance in cytoplasm. Regaud fixation, Cleveland and Wolfe stain.

5 An adjacent section through the same cell as that shown in figure 1. The number and distribution of mitochondria shown here cannot account for the granular character of this cell as it appears with the Cleveland and Wolfe stain. Regaud fixation, Fain and Wolfe mitochondrial stain.

6 Section through a sparsely granulated erythrosin cell similar to that shown in figure 2. Note dull blue color of cytoplasm as contrasted with figure 5. Regaud fixation, Fain and Wolfe mitochondrial stain.

7 An adjacent section through the same cell as that shown in figure 3, showing mitochondria. Regaud fixation, Fain and Wolfe mitochondrial stain.

8 An adjacent section through the same cell as that shown in figure 4. The mitochondrial pattern cannot acount for the reddish material found when the erythrosin technique is used. Regaud fixation, Fain and Wolfe mitochondrial stain.

9 Erythrosin cell as it appears with the Cleveland and Wolfe stain after sublimate-saline-formol fixation.

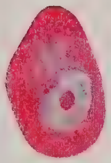
10 Adjacent section through the same cell as that shown in figure 9, stained selectively with azocarmine to show the identity of the erythrosin cell and the carmine cell. Sublimate-saline-formol fixation, Dawson and Friedgood stain.

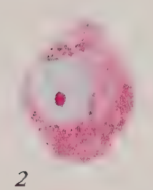
11 Typical orange cell. Sublimate-saline-formol fixation, Dawson and Friedgood stain.

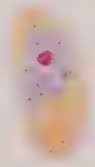
12 Partially degranulated basophile. Sublimate-saline-formol fixation, Dawson and Friedgood stain.

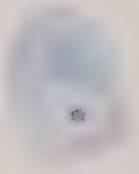
13 Basophile free of reddish substance, but showing some clumping of granules. Regaud fixation, Cleveland and Wolfe stain.

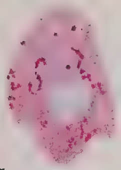
14 Erythrosin cell stained with orange G and aniline blue only. For explanation see text.

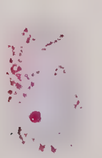

1

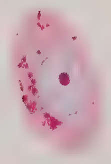

2

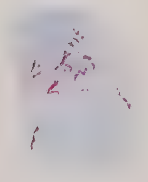

3

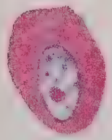

4

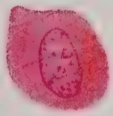

5

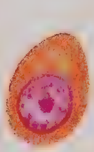

6

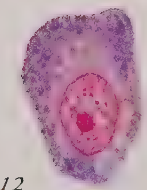

7

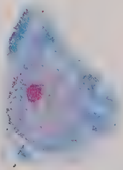

8

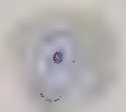

9


10


11


12


13


14

THE POSTPARTUM HISTORY OF THE CORPUS LUTEUM OF THE CAT

ALDEN B. DAWSON
The Biological Laboratories, Harvard Unverisity, Cambridge, Massachusetts

THREE PLATES (TWELVE FIGURES)

In an earlier paper (Dawson, 41a) the development and morphology of the corpus luteum of the cat during gestation were described but at that time only limited observations had been made on ovaries following parturition. More material is now available and it is possible to outline the general history of the corpus luteum in both lactating and non-lactating animals for considerable periods after delivery. However, data for the exact determination of the full life-span of the corpora are still lacking, but indirect and circumstantial evidence enable approximations to be made of the length of time that these bodies may persist in the ovaries. The corpora lutea of pseudopregnancy have not been studied beyond the twenty-seventh day after mechanically induced ovulation and they will therefore not be discussed in this paper.

MATERIALS

The ovaries that form the basis of this study were routinely removed from animals used in a study of the changes occurring in the pituitary gland during the reproductive cycle. They were usually fixed in Bouin's fluid, sectioned serially, 10 μ thick, and stained with hematoxylin and eosin. A few specimens were fixed in a Helly-osmic solution and later treated with osmic acid. The cats were obtained by purchase and their previous reproductive activity is accordingly unknown. Ordinarily the longest period that animals were kept under observation was from late December until the end of July, the normal breeding period for this latitude (Dawson, '41b).

DESCRIPTION

Corpora lutea of pregnancy

During pregnancy the corpora lutea of the cat undergo a rather remarkable series of histological changes. The follicular cells exhibit evidence of preluteal transformation as early as 6 hours after coitus and at 20 hours they appear as a lacy fringe of fibroblast-like cells lining the swollen follicle (Dawson and Friedgood, '40). They retain this pattern until the rupture of the follicle. Theca interna cells do not appear to undergo luteal transformation in the cat.

Immediately after ovulation the follicular cells become more compactly grouped and begin to hypertrophy and by the seventh day after mating they have acquired the polyhedral form typical of luteal cells. Growth of the cells continues until the twelfth to the sixteenth day when both the luteal cells and corporea reach their maximum size (fig. 1). At this stage the luteal cells are characterized by a fine, peripheral, uniform vacuolation. The unmodified cytoplasm occupies the center of the cell and contains the nucleus which usually assumes an eccentric location.

During the third week of pregnancy the fine peripheral vacuolation progressively disappears and the peripheral cytoplasm becomes irregularly fibrous (fig. 2). This transformation of the luteal cells is practically complete at the end of the fourth week and the histological picture persists for several weeks but it is gradually lost due to the reappearance of vacuolation. The second phase of vacuolation is well developed by the fiftieth day in all animals, but may be observed in some animals as early as the fortieth day, and continues until parturition. This second system of vacuoles is relatively coarse and the vacuoles completely fill the cytoplasm extending even to the surface of the nucleus. The vacuoles vary considerably in size and are ordinarily very numerous. However, scattered luteal cells may possess only one or a few large vacuoles. In extreme cases luteal cells may assume the form of a signet-ring characteristic of typical fat cells.

During the last days of pregnancy the luteal cells contain a variable number of small, refractile, acidophilic granules. They are sometimes aggregated in a paranuclear position but are frequently scattered throughout the cytoplasm of the intervacuolar septa.

Postpartum corpora lutea

There are few data for the cat on the fate of the corpora lutea after parturition, but the impression gained from the literature is that there is a very rapid involution of these bodies. Markee and Hinsey ('35) have described a case of probable superfetation in the cat. In this instance two kittens were delivered from the left uterine horn 13 days after a previous delivery of two kittens from the right horn. They state that two corpora were present in left ovary and none in the right, implying that complete involution of the corpora of the right ovary had occurred in the 13-day interval following the first parturition. This observation is not confirmed by the present study since persistent corpora have been found at relatively long periods after the termination of normal pregnancies.

Attention was first focussed on the corpora of the postpartum period by the finding of bright pink bodies in the ovaries of a cat that had been lactating for 9 days. In general appearance they resembled young corpora of the second week of pregnancy. However, circumstances precluded the interpretation of these bodies as the products of recent ovulations since all females in late pregnancy were isolated from the males and after delivery were kept in separate covered pens with their kittens. Furthermore the cat, unlike the rabbit, is known not to possess mature follicles at the time of parturition. As more data were accumulated, it became obvious that these conspicuous corpora were characteristic of lactating cats and could be recognized consistently in fresh ovaries until the end of the fourth week of lactation. The intensity of the color varied with the position of the individual corpus in the ovary; those in a superficial location being more conspicuous than those imbedded more deeply. Furthermore, the depth of the color of the corpora was progressively reduced after the sixteenth day of lactation, and in the later periods of lactation, beyond the fourth week, they were relatively inconspicuous. This behavior of the corpora in lactating cats was in striking contrast to that in non-lactating animals. When the kittens were not permitted to nurse, the postpartum ovaries appeared macroscopically as they did in late pregnancy and in the early days following parturition, with or without lactation. This differential behavior of the corpora, correlated with the maintenance or absence of lactation, may be recognized grossly as early as the ninth day but frequently does not become apparent until as late as the twelfth day.

Corporea lutea of lactation

During the first 4 days after delivery all corpora, both macroscopically and microscopically, appear unchanged. In some instances in lactating animals there is a clear increase in vascularity as early as the fifth day but this change may not occur until several days later. During early lactation the luteal cells are still vacuolated as in the last days of pregnancy and the cell size and nuclear size are comparable (fig. 3). Acidophilic granules are also present. Following the initial increase in vascularity there is a progressive disappearance of the smaller and more numerous vacuoles and there appears to be a considerable reconstitution of cytoplasm since there is no obvious reduction in cell size. There is, however, a tendency for the very large vacuoles to persist. This histological transformation of the corpora is usually complete by the twelfth day (fig. 5) and persists without great modification until the twenty-eighth day (figs. 6 and 7). Thereafter, there is a regression in the degree of vascularization and a concomitant increase in the number of large vacuoles in the luteal cells. There also appears to be a decrease in cell size. The refractile acidophilic granules, however, persist throughout the entire period and no significant changes in their number or distribution could be recognized. Observations on timed material were discontinued on the sixty-third day postpartum when the kittens were weaned (fig. 8).

Corpora lutea without lactation

So far as our observations extend, the corpora lutea of cats that were not permitted to nurse show little change in their histological pattern. The degree and type of vacuolation remain almost unchanged but there is gradual reduction in cell size. This indicates that there must be a slow and gradual removal of fat from the luteal tissue. Acidophilic granules are present but are very inconspicuous since their distribution is restricted to the thin intervacuolar cytoplasmic septa. Observations on the corpora of non-lactating animals were discontinued during the fourth week postpartum (fig. 4). At this time the follicles in the ovary had reached the proestrous level and the uterus had returned to the typical proestrous condition. The marked increase in vascularity observed in the corpora lutea of lactation apparently does not occur in the absence of active suckling by the young.

The probable life-span of the corpora lutea

Unfortunately interest in the postpartum history of the corpora lutea developed after the cat colony had been discontinued and during the war it became impossible to secure and maintain an adequate number of animals for a sufficient period of time to permit following the corpora lutea in timed material through their final stages of retrogression. However, it has been possible, from a study of all the ovaries in our collection, to gain a rather general picture of the stages of retrogression and also to determine within somewhat wide limits the presumptive life-span of the corpora. It has not been possible to determine if lactation is a significant factor in influencing the persistence of the corpora although the facts already recorded suggest that lactation tends to prolong the life of these bodies.

It is generally recognized (Dawson, '41b) that the breeding season of cats in this latitude ordinarily terminates in late July and that sexual activity is resumed in January. Accordingly a careful study was first made of the ovaries removed from anestrous and estrous animals in January, February and early March since the presumption was that any corpora present in these ovaries during these months would be those derived from a fertile mating of the preceding breeding season at least as late as July. If a successful mating had occurred even in early January these animals should have been pregnant since the normal period of gestation is 63 days. Admittedly the picture might be complicated by pseudopregnancy or early abortion. Correspondingly the ovaries of pregnant animals that were killed in February, March and April should yield equally valuable data if it could be established that two distinct classes of corpora were present. Likewise lactating animals of a slightly later period would be similarly important, provided two classes of corpora were also found. In both instances the presumption would be that the more recent corpora represented the first pregnancy of the present breeding season and that the older corpora had persisted from a mating at the end of the previous breeding season, probably not later than the last of July. Less valuable data should also be obtained from animals that were pregnant in July or August. In these cases the older corpora might be derived from a mating as early as January. Similarly anestrous or estrous animals of this period, lacking evidence of recent pregnancy or lactation, might yield old corpora from an earlier pregnancy.

Old corpora were found in anestrous animals killed on January 28, March 1, 2 and 27 and in estrous animals on November 24, December 4, January 25, February 1, March 2 and April 1. (The early estrus in November and December was induced by increased daily illumination of the animal quarters (Dawson, '41b). Two classes of corpora were found in pregnant cats in the early part of the breeding season on the following dates: February 27, seventh day of pregnancy;

February 10, tenth day of pregnancy; and March 27, fifty-sixth day of pregnancy, and in postpartum animals on March 24, 6 days postpartum; March 29, 4 days postpartum; April 25, 5 days postpartum, and April 27, 6 days postpartum. In addition, in the latter part of the breeding season two classes of corpora were found in lactating females on June 23, twelfth day, July 15, eighth and and July 24, fourth day of lactation respectively.

Single classes of old corpora in varying degrees of retrogression were also found in anestrous and estrous cats and in cats shortly after mating on the following dates: May 15, 24, 31 (2); June 1, 13 (2); July 2 and August 7. Since none of these animals gave any evidence of recent lactation it is apparent that successful mating must have occurred in the early part of the breeding season presumably not earlier than January but possibly somewhat later. Turner and deMoss ('34) state that following weaning, lactation is quickly inhibited, followed by a gradual involution of the lobule-alveolar system until at about 80 days only the duct system remains. No microscopic studies were made of the mammary glands of our animals. The condition of the glands was determined by palpation only.

It has already been established, in the study of timed material, that with lactation the corpora lutea may remain relatively well developed until the sixty-third day after parturition (fig. 8), approximately 126 days after mating. It now remains to determine the probable length of time that corpora may persist in the ovary. A corpus in the most advanced stage of retrogression that was found in this survey is illustrated in figure 12. The ovary was removed from a cat on March 29, 4 days after delivery. Two classes of corpora were present, the old type illustrated and younger corpora of the pregnancy just terminated. This was obviously the first pregnancy of the present breeding season. Accordingly, the old corpora must have been derived from a pregnancy late in the preceding breeding season, probably in July. Thus corpora in an advanced stage of retrogression have a probable age of about 8 months, calculated from the time of mating. This conclusion regarding the maximum life-span of corpora is further substantiated by similar calculations based on the somewhat fragmentary histories of the other animals previously listed as possessing old corpora. It seems unnecessary to present detailed analyses of each case.

The data on the length of life of the corpus luteum in other animals are not voluminous and in most instances the reproductive behavior of such animals is very different from that of the cat. The rabbit, like the cat, does not ovulate spontaneously but unlike the cat, the rabbit possesses mature follicles at the time of parturition and may become pregnant shortly after delivery. In both animals corpora lutea are formed only after successful mating or after artificial ovulation. According to Togari ('26) some corpora in the rabbit have wholly disappeared as early as 22 days after parturition while others persist for varying periods up to 55 days. The studies were apparently based on non-pregnant animals and no mention is made regarding the presence or absence of lactation. Hammond and Marshall ('25) report that in suckling rabbits a slight but irregular diminution in the size of the persisting corpora lutea of the previous pregnancy is found when they are compared with corpora of control animals from which the young were removed. Their observations were stopped on the fortieth day after parturition. At this time the corpora of suckling does were about one-half the size of those measured 4 days after delivery, while in control animals the glands were reduced one-third in size.

Retrogression of corpora lutea

Although direct evidence cannot be presented regarding the relative ages of the old corpora under study, an attempt has been made to arrange them in a series representing progressive stages of involution. Many corpora of a presumptive age of several months present the hepatoid appearance shown in figure 9. Vacuolation is reduced to a minimum and only vacuoles of a relatively large size are present. The cytoplasm appears uniformly granular and the brilliant acidophilic granules present in late pregnancy and during the first weeks of the postpartum period are absent. The small blood vessels including the capillaries have reduced lumina and are invested by a varying number of adventitial cells of unknown origin. They are not smooth muscle cells. Presumably they are derived from the capsular region and migrate centrally along the blood vessels. However, the possibility that they are detached endothelial cells should not be excluded (Corner, '45). No mitoses were observed in these accessory cells. The investing coats are always thicker at the periphery of the corpus but this may be correlated only with the greater size of the vessels in this area and may not be primarily indicative of a centripetal migration.

In still later stages the hepatoid appearance is retained and the accessory cells about the smaller units of the vascular system become more numerous. The luteal cells become somewhat smaller but still possess a uniformly granular cytoplasm with little or no vacuolation (fig. 10). As long as the hepatoid appearance is maintained there is usually no evidence of necrosis of the luteal cells. Only in one instance was any considerable destruction of luteal tissue observed. This was seen in an ovary removed from a cat on June 23 on the twelfth day of lactation. Here the hepatoid tissue was broken up into islets and irregular cords by fibroblastic invasion. Considerable numbers of large active macrophages containing a light brown pigment, vacuoles and ingested cell fragments were also present.

Evidences of any considerable necrosis of luteal cells ordinarily do not become evident until the investment of the blood vessels has advanced to a relatively high level and the adventitial cells have begun to lose their close, compact localization and to spread into the surrounding luteal tissue (fig. 11). At this stage the luteal cells are much smaller, and may exhibit some vacuolation. Isolated luteal cells then round up, their cytoplasm becomes intensely acidophilic and the nuclei become homogeneous and densely basophilic. Such nuclei may fragment and appear as small globules. This diffuse cytolysis appears to be progressive but never becomes sufficiently extensive to elicit any invasion of the involuting corpora by leucocytes or tissue macrophages.

As the luteal cells are reduced in number there is a concomitant spread of the adventitial cells from the loci representing the retrogressing vascular channels until only isolated luteal cells can be recognized (fig. 12). The adventitial cells are then spindle-shaped and a delicate network of collagenous fibers is developed. In these late stages the corpora have a characteristic pattern incompletely illustrated in figure 12. This pattern appears to be due to the radial distribution of the adventitial cells from the blood-vessel loci. In many instances these cells appear not to always radiate directly from a center but may follow curving pathways producing complex whorled or ''pin-wheel'' effects. The final stages in the transformation of a corpus luteum with the complete disappearance of luteal cells has not been observed, but a study of the late stages of involution suggests that ultimately the completely infiltrated corpora may be incorporated into the general interstitial tissue of the ovary.

The possibility that luteal cells might be directly transformed into spindle-shaped elements was tentatively considered but the diffuse cytolysis of luteal cells, observed in the later stages of retrogression, strongly indicates that they are eventually completely replaced by the infiltration of adventitial cells associated with the blood vessels. The exact site of origin of the adventitial cells cannot be determined. They may be of endothelial origin or derived from the thecal cells which in turn come from the general mesenchymal stroma of the ovary. In final analysis all the cellular components of the ovary come originally from the mesoderm and the extent to which the several cell types of the ovary may be interconvertible is an unsettled question (Corner, '45). This is strikingly illustrated in follicular atresia in the cat. In the atretic bodies the granulosa cells are not the dominant elements of the transforming follicle. They do not appear to be primarily involved in lipid metabolism and the follicular epithelium as such eventually disappears. The thecal cells, on the other hand, hypertrophy greatly and acquire a rich lipid content, usually distributed as small droplets of uniform size completely infiltrating the cytoplasm.

During its entire history each corpus ordinarily appears as a separate discrete body with a well defined capsule formed primarily by the theca interna of the mature ovarian follicle. In a few instances two closely adjoining corpora may be fused together to form a single mass and in exceptional cases superficial corpora may be everted and mushroom out on the surface of the ovary. However, they are readily recognized in all ovaries and even in late stages of involution may be distinguished easily from large atretic bodies which are lobulated and not definitely encapsulated (Kingsbury, '39).

The size of the corpora lutea

In general the size of the corpora lutea tends to vary directly with the size of the luteal cells of which they are composed. The number of cells remains relatively constant since there is little or no mitosis after the early formative period and the amount of stromal tissue, except in the later stages of retrogression, is relatively small in proportion to the amount of glandular parenchyma present. Nuclear size seems also to be correlated with cell size as can be readily seen from a study of the photomicrographs (figs. 1 to 12) which were taken at a uniform magnification. Both nuclei and cells are largest at the end of the growth period, 12 to 16 days after mating. In the succeeding period when the cytoplasm appears fibrous both cells and nuclei tend to be slightly smaller and the nuclei reach an almost minimum size in the last weeks of pregnancy when the cytoplasm becomes completely permeated with a system of relatively coarse vacuoles. Thereafter until the final stages of involution occur the nuclear size remains almost constant even when the luteal cells appear as isolated units among infiltrating spindle cells. There are, however, some unexpected variations in nuclear size in individual corpora lutea (see especially figs. 1 and 9). In some instances the volume of the nuclei appears to be doubled. In these cases large cell size tends also to be correlated with increased nuclear size. Occasionally large binucleated cells with nuclei of normal size are present (fig. 9). Such conditions suggest that at some time there has been a dissociation of the normal processes involved in mitosis, resulting in the failure of cytoplasmic division and permitting the reconstitution of the daughter chromosomes either into two equal nuclei or into one single large nucleus, a condition so commonly observed in the mammalian liver, especially of the rat (Dawson, '40; Beams and King, '42). It does not seem possible to interpret these great variations in nuclear and cell size as functional hypertrophy since they occur in cells of a common population in a single corpus where presumably the level of functional activity of the luteal cells is the same. However that there is a general correlation of nuclear and cell size with changing functional activity during the life of each corpus cannot be denied.

The chromatic patterns of the nuclei of the luteal cells also present some features of interest. In the early preluteal transformation that occurs in the preovulatory follicle, the nuclei of the granulosa cells show a reduction in the density of the chromatin network. They become vesicular and contain a prominent nucleolus. This condition of the nuclei reaches a maximum at the end of the growth period (fig. 1) and in the subsequent history of the luteal cells their chromatic network becomes denser but the nucleoli remains surprisingly well defined even in late stages of retrogression.

Vascularity

The vascular supply of the corpora lutea has been studied only in serial sections, without the aid of injection methods. In rapidly growing corpora the blood vessels, derived primarily from the theca interna, give rise to a rich bed of thin-walled capillaries composed almost entirely of endothelial cells with few adventitial cells. Later the larger, more peripheral vessels become more highly organized and the adventitial cells are more numerous. Regression of the capillary supply does not become obvious until the massive infiltration of luteal cells with fat, seen in the last weeks of pregnancy, has taken place. After parturition and without maintenance of lactation the corpora remain pale and the capillary bed remains restricted. However if lactation is maintained there is both an extension and engorgement of the vascular bed and the corpora becomes pink in color. Such changes are sometimes recognizable as early as the fifth day and are conspicuous by the twelfth day. Concurrently there is a loss of fat from the luteal cells and a reconstitution of the cytoplasm within them. It was felt that this might represent a rejuvenation of the luteal tissue under the influence of pituitary prolactin (luteotrophin) since there is a great reduction in the granulation of the pituitary carminophiles during this period (Dawson, '46). However, an examination of the uteri of such lactating cats gave no evidence of the production of progesterone. The uteri underwent progressive involution after the initial regeneration of the endometrium (unpublished data). On the other hand the absence of a progestational reaction in the uterus might have been determined by a lack or a very low level of estrogen at this time since there is little follicular development in the post-partum ovaries of lactating animals.

A much simpler explanation of the histological changes seen in the corpora of lactation might be based on the increased metabolic demands that are occasioned by the production of milk, the increased vascularity and depletion of the fatty reserves of the corpora being regarded as a response to the need of the organism for fats. However such small stores as are represented in the corpora would seem to be of little assistance in furnishing nourishment for the young.

Vacuolation

It is to be regretted that only a few ovaries were fixed and treated with osmic acid for the demonstration of lipids.

Accordingly most of the description of the luteal cells is based on material from which the lipids had been dissolved. However, from our knowledge of the appearance and distribution of lipids in the corpora lutea of other animals it seems safe to assume that the presence of well defined vacuoles in the luteal cells in indicative of the accumulation of lipids of some kind. It is also possible that early stages of the accumulation of lipids have not been recognized in the absence of adequate staining methods. Furthermore without selective staining methods it is impossible to determine which lipids are of physiological significance and which are the results of retrogressive changes.

The fine, peripheral vacuolation which occurs in the first 2 weeks of pregnancy doubtlessly represents lipid of functional significance since the condition of the uterus indicates that progesterone is being liberated under the stimulus of pituitary luteotropin (Dawson and Kosters,'44). The fibrous appearance of the luteal cells which follows this stage is difficult to explain. It may represent a peripheral canalization brought about by the rapid release of secretion. Corpora lutea are actively secreting at the time since ovariectomy from the eighteenth to the forty-fifth days of pregnancy is invariably followed by abortion (Courrier and Gros, '35, '36). Indirect evidence also suggests that during this period the secretory activity of the corpora may be maintained by placental luteotropin (Dawson, '46). In the final stages of pregnancy when the corpora are massively infiltrated with lipids, appearing as a coarse system of vacuoles, the secretory activity has apparently ceased since gestation proceeds normally when the ovaries are removed at any time after the forty-ninth day (Courrier and Gros, '35, '36). Whether or not the increased vascularization and withdrawal of lipids (loss of vacuolation) that occurs during the second week of lactation represents a mild reactivation of the corpora cannot at present be determined. There is no morphological or physiological evidence to support such an interpretation.

Just how much significance can be attached to variations in vacuolation of the luteal cells is problematical. In the marten highly vacuolated luteal cells, comparable to those in the cat in late pregnancy, are present during the long period in which blastocysts remain unimplanted in the uterus (Wright, '42). It seems probable that the corpora in the marten would undergo some regenerative change before implantation takes place, although Wright has not studied ovaries after implantation had occurred. Thus it appears that fatty infiltration of luteal cells, at least in exceptional cases, may be associated with temporary cessation of secretory activity and are not always indicative of irreversible retrogressive changes.

SUMMARY

The histological appearance of the postpartum corpora lutea of the cat is described and compared with that of the corpora lutea of pregnancy. The corpora lutea of lactating animals become bright pink at the end of the first week after parturition and retain this color until at least the end of the fourth week. The color seen in fresh ovaries is due to an increase in vascularity and is associated with a rather rapid disappearance of the smaller vacuoles which are seen to permeate the luteal cells in late pregnancy and in the early postpartum period. Some of the large, ''giant'' vacuoles persist. Observations on timed material were discontinued at the ninth week of lactation.

These changes in the corpora were not observed in females from whom the kittens had been removed at birth. The characteristic vacuolation persists but the cells gradually decrease in size, indicating that there is gradual withdrawal of fatty material. Dated material up to the fourth week after parturition was available for study.

The functional capacity of the apparently rejuvenated corpora of lactation is not known, but it is suggested that while the released pituitary prolactin (luteotropin) has its primary effect on the mammary gland a secondary effect is also produced in the luteal tissue. However, a study of the uteri of

lactating animals gave no direct evidence of the release of progesterone. The absence of a uterine response may be explained by the lack of sufficient estrogen to condition the uterine tissue properly.

Indirect and circumstantial evidence make it appear likely that the total life-span of the corpora, dated from the time of mating may be as long as 6 to 8 months. Two classes of corpora have been found in ovaries of pregnant and lactating animals both in the early and late portions of the breeding season which usually extends from January to July. It is assumed, accordingly, that the older corpora found in the early breeding season probably represent ovulations that occurred in the previous July while those of the late breeding season may be derived from ovulations which occurred as early as January of the current breeding season.

The process of involution has been followed and it was found that eventually the luteal tissue was reduced by a diffuse but progressive cytolysis which is usually not accompanied by any invasion of leucocytes or tissue macrophages. Adventitial cells, associated with the luteal blood vessels, gradually infiltrate the luteal parenchyma to replace the degenerating luteal cells. The ultimate source of these infiltrating cells has not been determined.

A comparison of the changes occurring in the corpora of lactating and non-lactating animals suggests that lactation may prolong the life of the corpora but in the absence of timed material for the late stages of retrogression no definite conclusion can be drawn.

LITERATURE CITED

Beams, H. W., and R. L. King 1942 The origin of binucleate and large mononucleate cells in the liver of the rat. Anat. Rec., vol. 83, pp. 281–297.

Corner, G. W. with the collaboration of C. G. Hartman and G. W. Bartelmez 1945 Development, organization, and breakdown of the corpus luteum in the rhesus monkey. Carnegie Inst. Wash. Pub. 557, Contrib. to Embryol., no. 204, pp. 117–146.

Courrier, R., and G. Gros 1935 Contribution à l'endocrinologie de la grossesse chez la chatte. C. R. Soc. Biol., vol. 120, pp. 5–7.

———— 1936 Dissociation foeoplacentaire realisée par la castration chez la chatte. Ibid. vol. 121, pp. 1517–1520.

DAWSON, A. B. 1940 Cell division in relation to differentiation. Growth (suppl.), pp. 91–107.

——— 1941a The development and morphology of the corpus luteum of the cat. Anat. Rec., vol. 79, pp. 155–169.

——— 1941b Early estrus in the cat following increased illumination. Endocrinology, vol. 28, pp. 907–910.

——— 1946 Some evidences of specific secretory activity of the anterior pituitary gland of the cat. (Accepted for publication, Am. J. Anat.)

DAWSON, A. B., AND H. B. FRIEDGOOD 1940 The time and sequence of preovulatory changes in the cat ovary after mating or mechanical stimulation of the cervix uteri. Anat. Rec., vol. 76, pp. 411–429.

DAWSON, A. B., AND B. A. KOSTERS 1944 Preimplantation changes in the uterine mucosa of the cat. Am. J. Anat., vol. 75, pp. 1–27.

HAMMOND, J., AND F. H. A. MARSHALL 1925 Reproductions in the rabbit. Edinburgh.

KINGSBURY, B. F. 1939 Atresia and the interstitial cells of the ovary. Am. J. Anat., vol. 65, pp. 309–331.

MARKEE, J. E., AND J. C. HINSEY 1935 A case of probable superfetation in the cat. Anat. Rec., vol. 61, pp. 241–251.

TOGARI, CH. 1926 On the corpus luteum of the rabbit. Folia. Anat. Jap., vol. 4, pp. 337–362.

TURNER, C. W., AND W. R. DEMOSS 1934 The normal and experimental development of the mammary gland. I. The male and female domestic cat. Mo. Agr. Exp. Sta. Res. Bull. 207, pp. 5–15.

WRIGHT, P. L. 1942 Delayed implantation in the long-tailed weasel (Mustela frenata), the short-tailed weasel (Mustela cicognani) and the marten (Martes americana). Anat. Rec., vol. 83, pp. 341–353.

EXPLANATION OF PLATES

All figures are photomicrographs of corpora lutea, taken at a uniform magnification (× 460). All material was fixed in Bouin's fluid and stained with hematoxylin and eosin.

PLATE 1

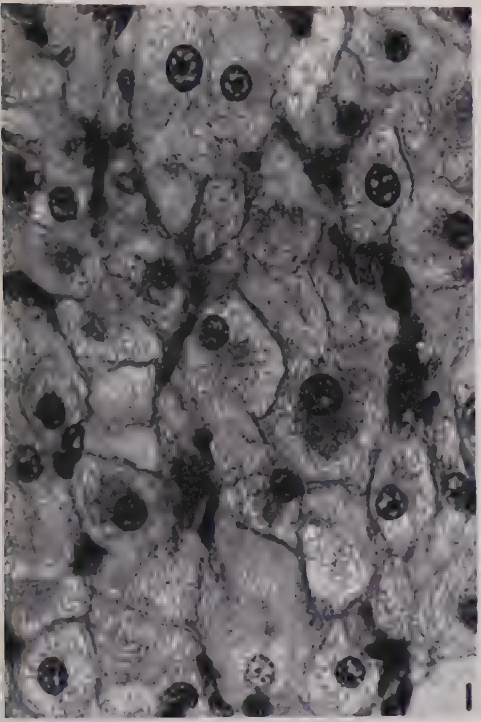
EXPLANATION OF FIGURES

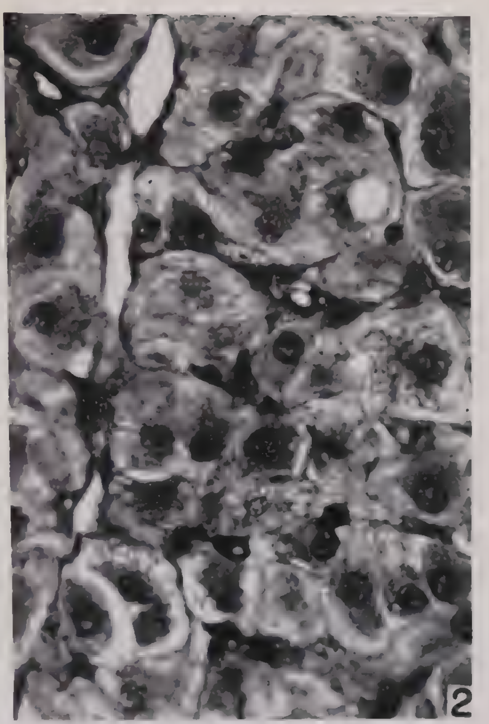
1 Corpus luteum, 14 days after mating, showing the extremely fine peripheral vacuolation and in some instances the denser paranuclear cytoplasm. Both cells and nuclei are at a maximum size. The interstitial tissue is scanty and the capillaries have few adventitial cells associated with them.

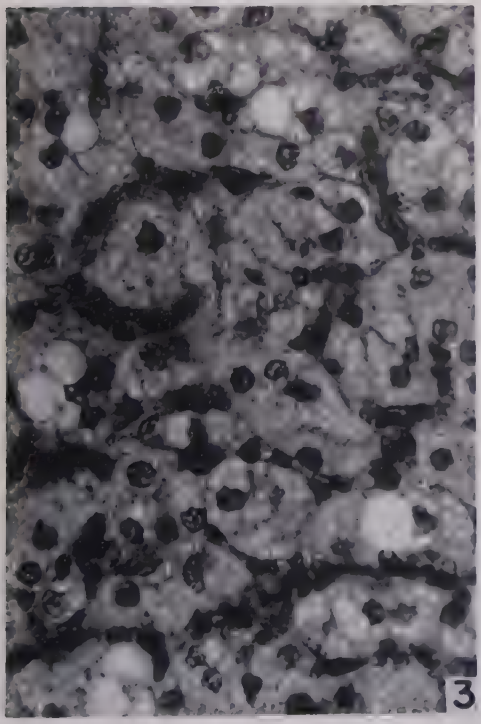
2 Corpus luteum, 21 days after mating, showing the replacement of the uniformly fine, peripheral vacuolation of the luteal cells by an irregular fibrous appearance.

3 Corpus luteum, 5 days after parturition, with lactation, showing the majority of the luteal cells almost completely filled with numerous relatively large vacuoles. Less commonly the cells may contain one to several very large vacuoles. The cell outlines are not always distinct but the relative size of the cells can be determined from the spacing of the nuclei. Large capillaries are conspicuous.

4 Corpus luteum, early in the fourth week postpartum, without lactation, showing the closer spacing of nuclei indicative of a reduction in volume of the luteal cells. The vacuolation is of the same order as in figure 3, though less regular.


1


2


3

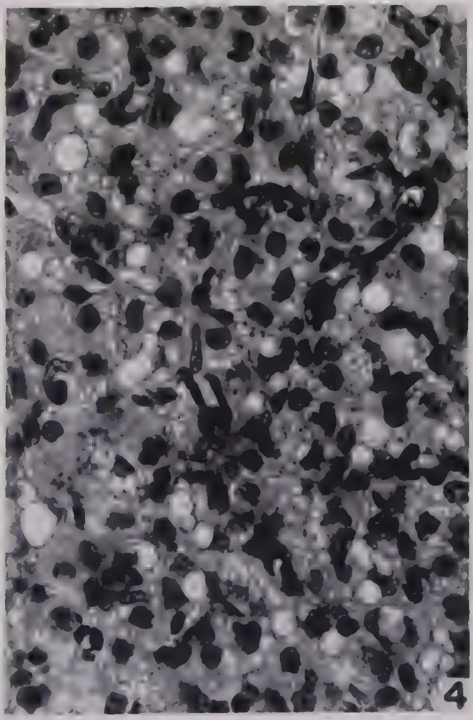

4

PLATE 2

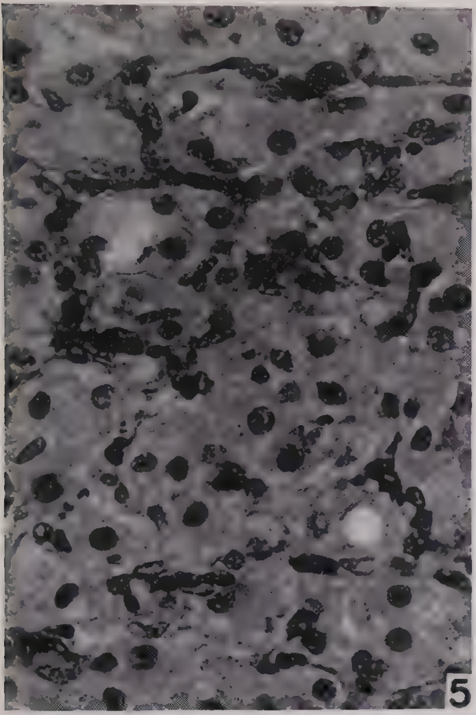
EXPLANATION OF FIGURES

5 Corpus luteum at the twelfth day of lactation (four kittens) showing the almost complete disappearance of the uniform vacuolation so characteristic of late pregnancy and early lactation. Persisting vacuoles are chiefly of the large solitary type. The cytoplasm contains scattered, refractile acidophilic granules. There is an increase in vascularity, in keeping with the red color of the corpora in the fresh ovary.

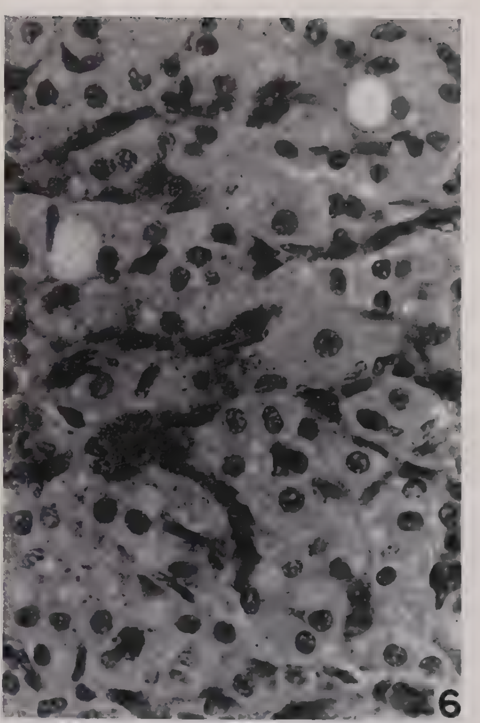
6 Corpus luteum at the seventeenth day of lactation (two kittens) showing a condition comparable to that of the twelfth day.

7 Corpus luteum at the twenty-eighth day of lactation (four kittens, 4 days; two kittens, 24 days). There is an increase in vacuolation.

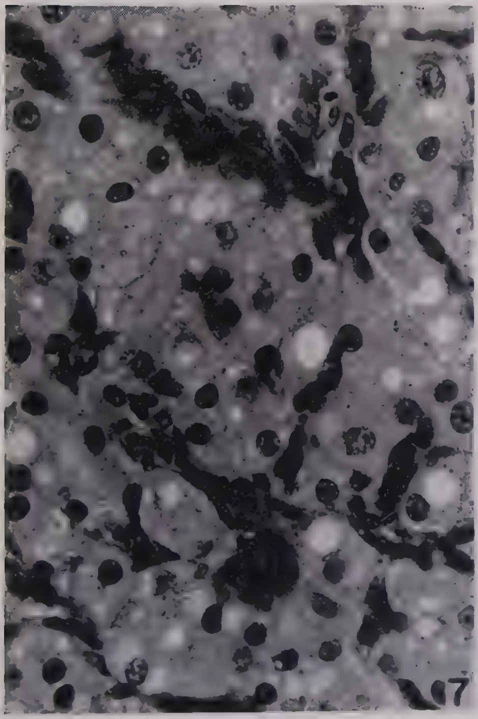
8 Corpus luteum at the sixty-third day of lactation (three kitens). The vacuolation is more extensive and the cells are more irregular in size.



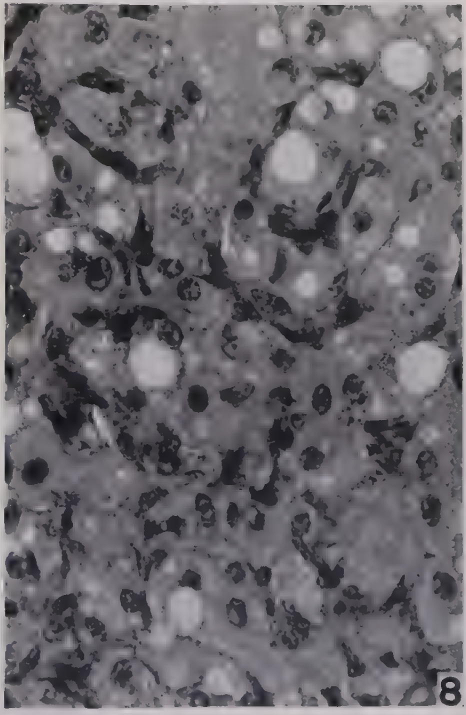
5



6



7



8

BOSTON MEDICAL
FEB 1
LIBRARY

PLATE 3

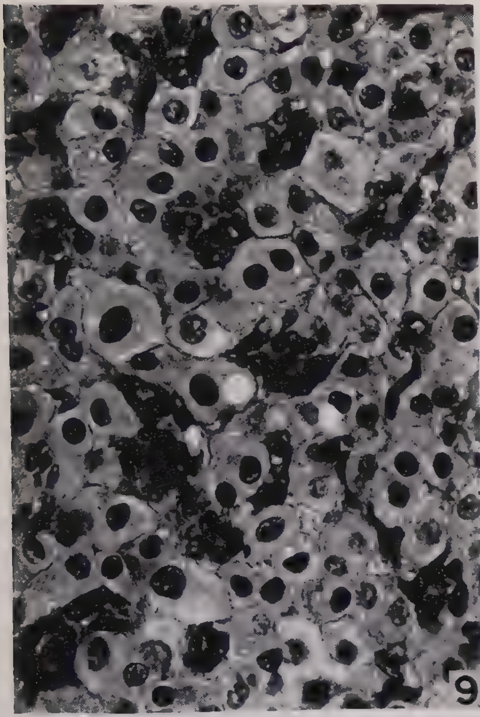
EXPLANATION OF FIGURES

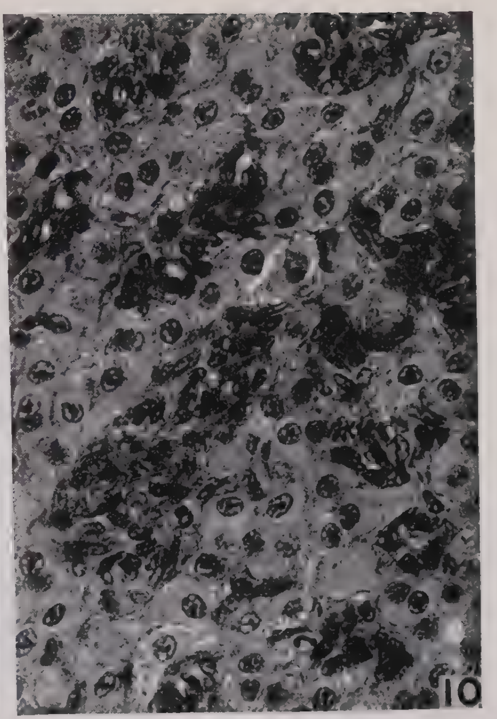
9 Corpus luteum from an anestrous cat of unknown history, killed on August seventh. No positive evidence of recent lactation; four corpora of same type in right ovary, none in the left. Greatest possible age of corpora is 7 months, possibly from a pregnancy beginning in early January. Vacuolation was scanty in all corpora. Both cells and nuclei are of variable size with occasional binucleated cells. Capillaries are rather heavily invested by adventitial cells.

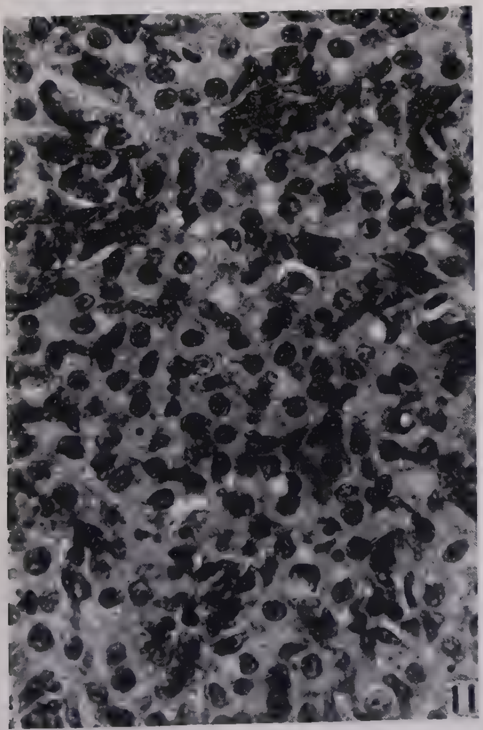
10 Corpus luteum from an estrous cat killed on June 28 (1 hour and 40 minutes after mating). Prior breeding history was not known. Earliest previous pregnancy could have occurred in early January. The adventital cells about the capillaries are conspicuous and vacuolation of luteal cells is almost completely absent. The cytoplasm appears uniformly finely granular.

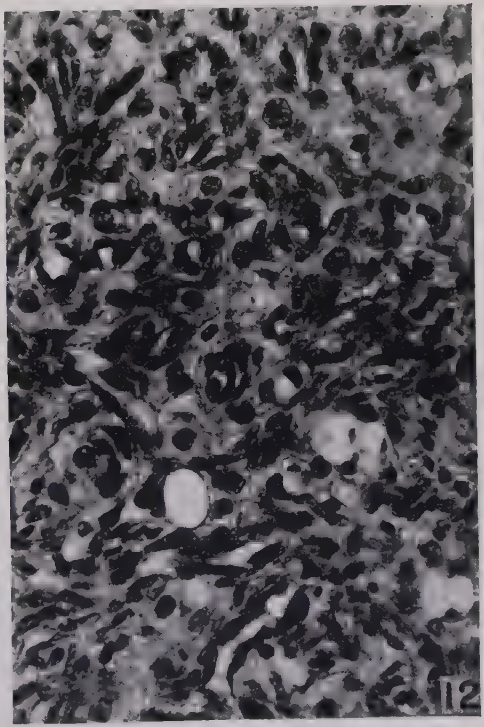
11 Corpus luteum from an estrous cat, mated on May 23 and killed 29 hours later. This animal was received April 18 in an anestrous state without any evidence of either recent pregnancy or lactation. The presumption is that the corpora had persisted from the previous breeding period which usually ends in July. The luteal cells are small and exhibit varying degrees of vacuolation. Occasional cells appear to be degenerating. The adventitial cells about the compressed capillaries have begun to infiltrate the surrounding compact luteal tissue. There is no invasion of leucocytes or wandering cells.

12 Corpus luteum from a cat, 4 days postpartum, whose kittens were not permitted to nurse. Date of parturition, March 25. Two classes of corpora lutea were present, the old type illustrated and the younger corpora of the pregnancy just terminated. The presumption is that this is the first pregnancy of the present breeding season and that the old corpora are derived from a late pregnancy of the preceding breeding season. The luteal tissue appears to be massively invaded by spindle-shaped cells which radiate irregularly from scattered loci suggesting their derivation from the adventitial cells associated with the small blood vessels. Scattered typical luteal cells are still present; some vacuolated, others with refractive acidophilic granules.


9


10


11


12

AN ARTERIAL ANOMALY IN THE FETAL PIG[1]

ROBERT E. GREGG
Department of Biology, University of Colorado, Boulder

FOUR TEXT FIGURES AND ONE PLATE (ONE FIGURE)

Numerous malformations of the principal arteries of the human body have been described, and these have been summarized by Poynter ('16) in a monographic treatment. Poynter states (pp. 41–42) that,

> Some of these conditions are understood embryologically, but many of them are still without satisfactory explanation from a developmental standpoint . . . The arrangement of branches in many of these [cases] is similar to type forms in other mammalia. It is interesting to note that through all classes of mammals there is variation in the arrangement of branches from the arch in the individual species. No single species or genus shows a wide variation till primates are reached, and man seems to present as anomalous development all of the conditions encountered in other mammalia.

Seventeen of the more common anomalies of the human aorta and its branches have been figured by McDonald and Anson ('40), and of these, the seven most frequent types are herewith reproduced from their paper. (See figs. 1 and 2). It will be noted (fig. 1), that the normal arrangement (A) includes three branches from the aortic arch, (1) the innominate on the right (left in ventral view), from which arise the right subclavian and the right common carotid, (2) the left common carotid, and (3) the left subclavian artery. Fairly frequent modifications of this pattern are seen (B) in which the left vertebral springs independently from the aortic arch just proximal to the left subclavian; (C) in which the innominate and the left common carotid are united at the base; and (D) in which the derangements observed in the two foregoing cases are exhibited in one specimen. These are all relatively minor changes. In figure 2 (E) the separate left vertebral has shifted to a position distal to the corresponding subclavian, and a few cases are known (F) in which the common carotids have each united with the corresponding subclavians. A very interesting origin for the right subclavian (G) has been reported in quite a number of instances. In these the anomalous vessel leaves the systemic aorta distal to the origin of the left subclavian, passes dorsal to the esophagus, crosses

---

[1] I wish to acknowledge assistance from the following persons: Dr. Carl R. Moore, Dr. L. J. Wells, Dr. E. G. Butler, and Dr. B. J. Anson. Their interest and suggestions have been greatly appreciated.

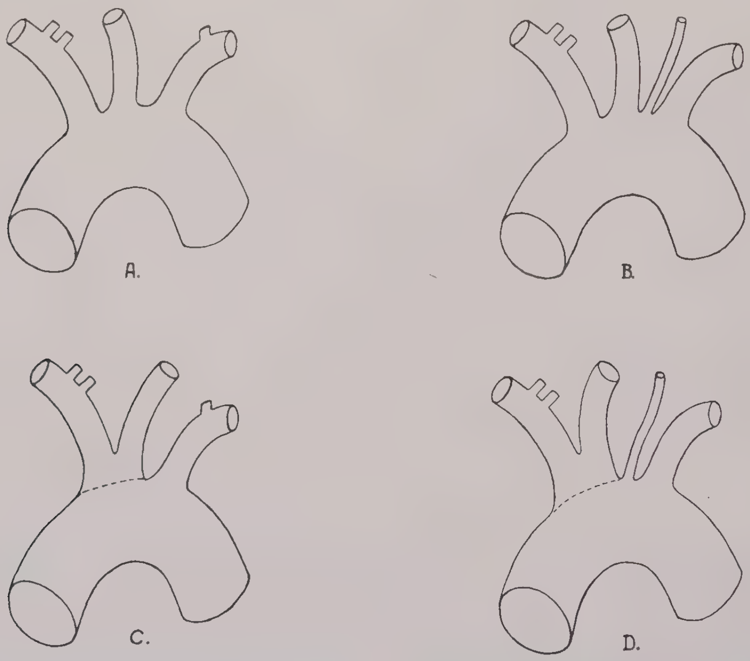


Fig. 1 Aortic anomalies reproduced from McDonald and Anson ('40, p. 96). A, normal arrangement of vessels of the human aortic arch. B, C, and D, commonest types of anomalous arrangement.

to the shoulder of the right side, and thence as the brachial to its usual distribution in the right arm.

This latter abnormality has been adequately explained as the result of rather obvious changes in the primitive aortic arch pattern. In the transformation of these arches, it is usual for the base of the right subclavian to originate from the persistent right fourth arch and most of the right dorsal aorta from this point posteriorly to the seventh intersegmental artery, which contributes to the axillary portion of the subclavian. The distal segment of the dorsal aorta of the right side normally degenerates and frees the subclavian from any attachment with the aorta so far caudad (fig. 4). On the contrary, the configuration shown in G has in all probability been formed following the functional retention of the above

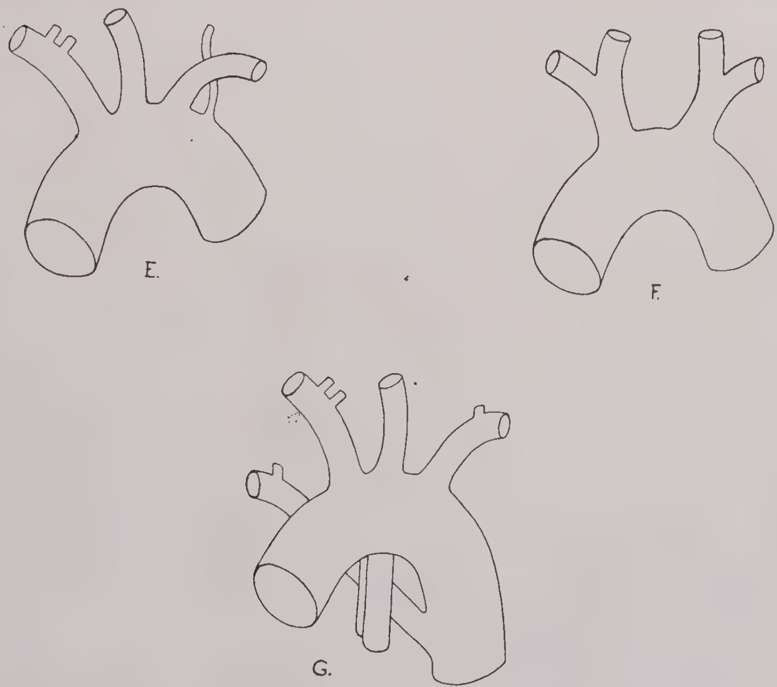


Fig. 2 Aortic anomalies reproduced from McDonald and Anson (l.c. p. 97). E, F, and G, somewhat less common types of abnormal arrangement.

mentioned distal portion of the right dorsal aorta, with a concomitant loss of the right fourth arch and its adjacent proximal dorsal aorta. The vertebral arose from the subclavian in normal fashion, from the fusion of the anterior intersegmental arteries. In some instances, however, the vertebral may arise independently from this retro-esophageal subclavian, and be attached to the right common carotid instead. This has been attributed to the use of the right fourth arch and its dorsal aorta for the basal portion of the vertebral, and the distal section of the aorta for that of the subclavian, with a break developing between them. This would

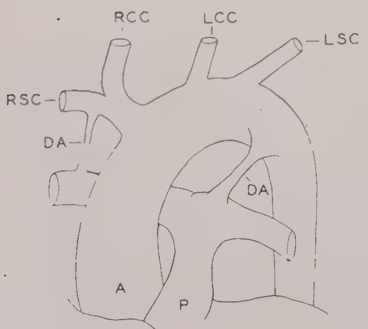


Figure 3

Fig. 3 Persistent double pulmonic arches reproduced from Poynter's monograph, figure 30. A, aortic trunk. DA, ductus arteriosus. LCC, left common carotid. LSC, left subclavian. P, pulmonary trunk. RCC, right common carotid. RSC, right subclavian.

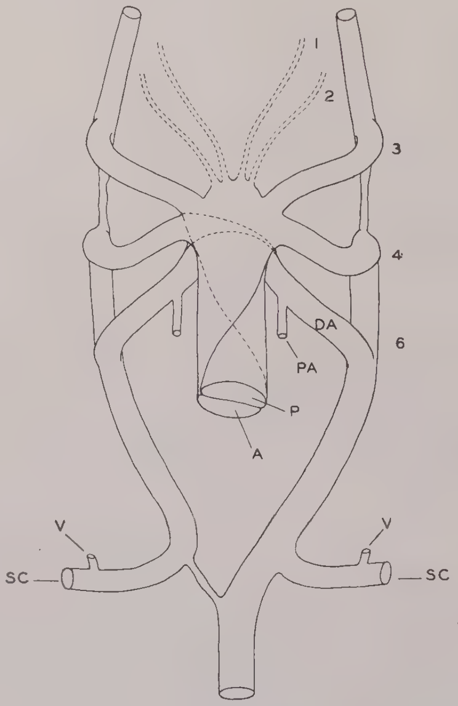


Figure 4

Fig. 4 Post-branchial phase of aortic arch transformations (modified from Arey, '31, after Congdon). 1, 2, 3, 4, and 6 (pulmonary) aortic arches. A, aortic trunk. DA, ductus arteriosus. P, pulmonary trunk. PA, pulmonary artery. SC, subclavian. V, vertebral.

necessitate the retention of the proximal portion of the sixth intersegmental as the connection between the right dorsal aorta and the developing vertebral.

Cairney ('25, p. 265), states that a retro-esophageal right subclavian has been observed in the rabbit by W. R. Smith (1891), in one out of 700 specimens examined.

The anomalous artery of the present report was discovered in a pig fetus 21 cm in length, and therefore one approaching fairly close to term. (Field, '39, p. 18, states that the gestation of the pig is 17 weeks, and that at birth the animal is 12 inches (30 cm) or more in length). The specimen is in the embryological collection of the University of Colorado. The right subclavian artery arises as a side branch of the pulmonary trunk on its dorso-lateral surface, and progresses dorsal to (beneath) the systemic aorta and directly to the region of the right shoulder where it turns outward as the brachial into the foreleg (fig. 5). The artery passes ventral to the trachea in contrast to most cases of anomalous subclavians attached caudally on the aorta which pass in a retro-esophageal course to the arm. The usual arterial branches, such as the costo-cervical, the thyro-cervical, the vertebral, and the internal mammary appear in normal position, and the right recurrent laryngeal nerve passes around the subclavian to run forward to the larynx. The pulmonary arteries, the ductus arteriosus, the bicarotid trunk, and left subclavian are all normal in position and distribution for the species. A fine wire leader with a blunt head was inserted through an incision on the ventral wall of the pulmonary trunk and passed into the aperture of the right subclavian where it joined this trunk, thus ascertaining the patency of the connection between the two vessels and establishing the course of blood flow as surmised from an inspection of the outside surfaces of these tubes. The wall of the pulmonary trunk was also incised longitudinally, and it was determined beyond all doubt that the right subclavian had an open connection with it, and the ductus and common pulmonary artery likewise. The ductus arteriosus has a smaller caliber than the pulmonary trunk, but this seems to be quite normal as judged by other specimens having normal vessels. If it is at all reduced in size, this might be traced to the diversion of blood in the pulmonary into the right subclavian in the case at hand.

The condition of the heart was found, by incision into all four chambers, to be normal. The right atrio-ventricular valve was tricuspid and the left atrio-ventricular valve was bicuspid, as in the normal heart, indicating no situs transversus in connection with the heart chambers. The semilunar valves of both sides showed three leaflets each, and the interventricular septum was complete indicating no mixing of aerated with non-aerated blood could have occurred, after birth, from this locus. The foramen ovale was broadly open between the two auricles, and no unusual features were noticeable in other portions of the heart.

The physiological consequences of the arrangement of blood vessels in this pig are interesting to ponder. It is obvious that no untoward effects are to be seen in the embryo as it has progressed almost to birth with no interference in the development of any of the regions of the body. Aerated blood from the placenta enters the fetal circulation and mingles with that in the general fetal flow. All tissues of the embryo, therefore, receive only mixed blood in contrast to the condition after birth, inasmuch as the lungs of the organism are not functional prior to parturition. As a result, it matters not from which cardiac vessels the blood comes in its distribution to the embryonic tissues since all is mixed and carries at least some oxygen. At the moment of birth extensive rerouting of the fetal blood is suddenly effected. Expansion of the lungs draws blood into the pulmonaries and leaves only a small amount to flow through the ductus for a limited period. Return of aerated blood from the placenta is, of course, permanently prevented. The foramen ovale closes producing a double circulation, one through the right heart and lungs, and one through the left heart and to the general systemic circulation.

Thus it is seen that no interference with oxygenation of the tissues is to be sought in this fetus, but it seems quite certain that such interference would have appeared immediately after birth. We can only speculate, but the conclusions rest at least on a firm anatomical foundation if not on observed physiological events. Since all the connections as described above were patent, we are not confronted with atresic conditions and interruption of blood flow. No direct anastomoses were demonstrable between the aorta and the right subclavian, so it is assumed that only non-aerated blood could enter the right subclavian, and hence the area supplied by it. The ductus arteriosus might have remained open providing a path for some aerated blood to the subclavian, but the back pressure from the aorta through this vessel and into the subclavian would not appear to be sufficient to account for it. The flow into the subclavian from the pulmonic trunk and the increased flow into the pulmonaries following the expansion of the lungs would seem to preclude any appreciable suction of blood from the aorta across the ductus. The ductus therefore would be expected to close. Passage of aerated blood through the left vertebral to the basilar and caudad through the right vertebral to the right subclavian would necessitate complete reversal of flow in the right vertebral. This must be utterly impossbile in the face of established pressures and directions of flow in the cranial arteries. If all these suppositions are correct, we are left the alternative of only non-aerated blood flowing into the right subclavian and to the shoulder and foreleg. Whether the small amount of oxygen remaining in so-called non-aerated blood would be sufficient to nourish these regions and protect the tissues from death is very doubtful; they would at best show an impoverished growth and be shriveled in size. More likely, the situation would lead to rapid gangrene of the foreleg and death of the entire organism from the resulting toxemia.

The extensive account by Poynter concerning anomalies of the human aortic arch and its branches was consulted for cases like the one herein described. Nothing identical was found, but two cases were described and figured (Poynter, pp. 39–40; figs. 15 and 16), which are quite similar in that a systemic vessel was connected solely with the pulmonary circuit! In these instances it was the systemic aorta arising as a direct continuation of the left pulmonary arch, the ductus arteriosus to be exact. The segment of the dorsal aorta on the left side between the fourth and pulmonary arches, and the fourth arch itself, atrophied in one case. In the other case the fourth arch formed the base of the left subclavian, but in each the systemic trunk had no connection with its original ventral aorta and the left heart. As a consequence, no aerated blood could be distributed to regions below the level of the heart, since the descending aorta received blood only from the pulmonary, unless the interventricular septum in these cases was incomplete, allowing a mixture of blood from the return systemic circulation with aerated blood from the lungs to pass into all arteries leaving the heart. The condition of the interventricular septum in these cases was not stated, but the diagrams indicate that the aortico-pulmonary septum in the original bulbus was complete, and that the two vessels connected with their corresponding heart chambers which were likewise separate. The physiological behavior in these anomalies was not discussed.

Persistent double pulmonic arches are described by Poynter (p. 23 and fig. 30, as originally recorded by Breschet, 1826), as a very rare phenomenon. The right ductus arteriosus extends anteriorly from the right pulmonary to the subclavian of the same side, and a similar relation obtains between the left pulmonary, the left ductus arteriosus, and the systemic aorta. The embryological interpretation is, obviously, a retention of the distal portions of the pulmonary arches in a functional though rudimentary condition (fig. 3).

Thus in human anatomy and from other animals there has been reported no abnormality that parallels the type presented in this paper. Those described from Poynter's work in the preceding paragraphs seem to be the closest approaches to it. The developmental explanation is not at once apparent, nor are my suggestions presented as the final and only possible solutions. Others may succeed in providing a clearer understanding when more embryological and comparative anatomical evidence is gathered. If we follow the theory of Baader (1886, from Poynter), that the aortic vessels and branches develop from a plexus of anastomosing channels, it is simple to derive any conceivable combination of connections resulting from increased flow and pressure in some of the tubes and decreased flow accompanied by obliteration in others. Such an explanation is plainly out of line with the facts of aortic arch development and transformation as understood in modern embryology.

Under normal conditions the spiral septum (aortico-pulmonary septum), dividing the truncus arteriosus (communis), into definitive aorta and pulmonary trunk, develops from the distal to the proximal end of the truncus and describes a spiral course. At the distal end the aortic portion of the tube is ventral in position and the pulmonary is dorsal. At the proximal end the relations are reversed, and the spiral septum unites with the interventricular in such a way that the aortic vessel communicates with the left ventricle and the pulmonary with the right ventricle. The spiral turn is in a clockwise direction and normally traverses 135 degrees. Various degrees of failure of the spiral septum to develop are known including complete absence, and in a similar manner, varying degrees of non-development of the interventricular septum leave the ventricles connected.

In figure 4 we have represented the post-branchial phase of aortic arch transformation modified to serve two possible interpretations of the anomalous right subclavian artery. A dotted line connects the anterior margins of the pulmonary or sixth arches and corresponds to the sulcus on the external dorsal surface of the aortic sac from which, on the inside, the spiral septum arises to grow backward as two intra-truncus cushions. Its spiral path is indicated also. It is possible that this took place in the usual fashion, but that the right sixth arch (right ductus arteriosus), beyond the pulmonary artery of that side has persisted as the main proximal channel for the right subclavian, with a concurrent occlusion and disappearance of the right fourth arch and the dorsal aorta between the fourth and sixth arches. The shifting of vessels during development (as demonstrated by Gladstone and Wakeley, '15), could easily account for the union of the pulmonary arteries into one tube where they join the pulmonary trunk, and this single tube is normal for the pig anyway (see fig. 5). The usual degeneration of the distal segment of the right dorsal aorta would separate the subclavian from the main aortic trunk and leave it joined only with the pulmonary trunk as figured. The alternative explanation involves an anomalous position of the external dorsal sulcus described above. It is conceivable that the sulcus and its internal connection with the spiral septum could have extended from the space between the fourth and sixth arches on the left side to the space between the fourth and third arches on the right (see upper dotted line). In this way the aortic and pulmonary trunks would be divided completely, and the basal portion of the right subclavian (4th arch and dorsal aorta) would be joined to the pulmonary instead of to the systemic aorta through the innominate artery as in the normal animal. The right sixth arch would degenerate.

Although this latter postulate requires a new position for the growing aortico-pulmonary sulcus, which has not been demonstrated and for which there have been established no precedents, it seems a not unlikely occurrence in view of the extreme anomaly we wish to understand. It would not presuppose a drastic shift in the relationships of primitive vessels, yet would accomplish a very drastic change in position of major adult vessels. The fact that the recurrent laryngeal nerve passed around the right subclavian in its normal position favors the retention of the fourth arch as the base of this artery. Had the sixth arch been responsible for the basal section of the subclavian, it is possible the recurrent laryngeal nerve would have a slightly more posterior position nearer to its partner of the left side which loops around the ductus arteriosus. As the heart sinks posteriorly into the thoracic cavity, however, it exerts such a drag on the arteries fastened in the cervical region that their positions are markedly changed as compared with those assumed during the postbranchial stages of the younger embryo, and it becomes very difficult to decide in this instance whether the fourth or the sixth arch has been retained. The location of the subclavian ventral to the trachea could have occurred regardless of which interpretation we adopt. In any event, we are faced with a problem that can be dispelled by recourse to the primitive phases of aortic arch transformation, but which cannot be solved in the present state of our knowledge. Unfortunately, there were no atrophic blood vessels in positions corresponding to undoubted fourth or sixth arches on the right side to aid in clearing up the early history of the embryo; the right subclavian appeared to have no connections other than that with the pulmonary trunk.

LITERATURE CITED

Arey, L. B. 1931 Developmental anatomy, a textbook and laboratory manual of embryology. Philadelphia, W. B. Saunders, 563 pp.

Baumgartner, W. J. 1924 Laboratory manual of the foetal pig. New York, Macmillan, 57 pp.

Cairney, J. 1925 The anomalous right subclavian artery considered in the light of recent findings in arterial development. J. Anat., vol. 59, pp. 265–296.

Congdon, E. D. 1922 Transformations of the aortic arch system during the development of the human embryo. Contrib. to Embryol., no 68, Carn. Inst. Wash. Publ., vol. 15, pp. 47–110.

De Garis 1932 Aortic axillary collaterals and the pattern of arm arteries in the anomalous right subclavian artery. Am. J. Anat., vol. 51, pp. 189–213.

Field, H. E. 1939 The fetal pig, an introduction to mammalian anatomy. Stanford Univ. Press, 53 pp.

Gladstone, R. J., and C. P. G. Wakeley 1915 Two cases considered from the developmental standpoint, in which the right subclavian artery arose from the arch of the aorta beyond the origin of the left subclavian. J. Anat. Physiol., vol. 49, pp. 362–375.

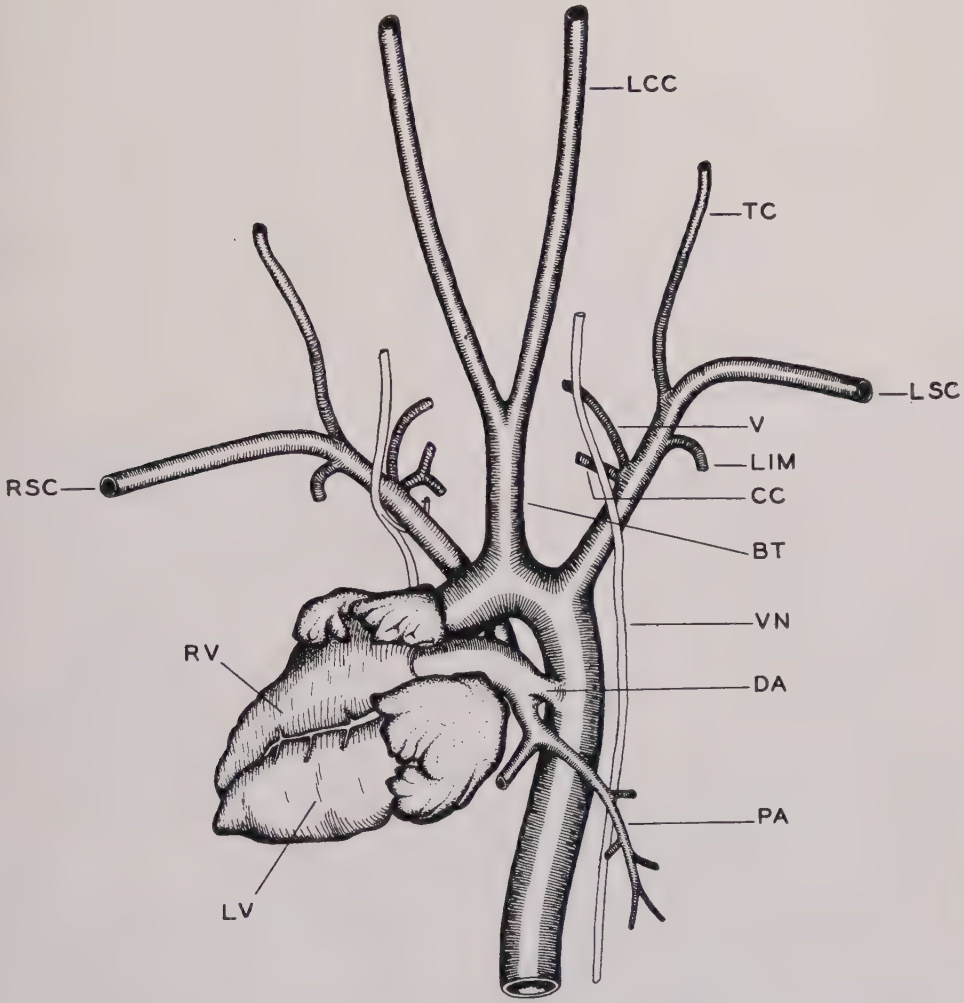
Hammer, D. L., and A. M. Meis 1941 Thyroid arteries and anomalous sub clavian in the White and the Negro. Am. J. Phys. Anthrop., vol. 28, pp. 227–237.

McDonald, J. J., and B. J. Anson 1940 Variations in the origin of arteries derived from the aortic arch, in American Whites and Negroes. Am. J. Phys. Anthrop., vol. 27, pp. 91–108.

Poynter, C. W. M. 1916 Arterial anomalies pertaining to the aortic arches and the branches arising from them. Univ. Nebraska Stud., vol. 16, pp. 229–345.

Smith, W. R. 1891 An abnormal arrangement of the right subclavian artery in the rabbit. J. Anat. Physiol., vol. 25, p. 325.

Wells, L. J. 1945 A case of iliothoracopagus (dicephalus tribrachius tripus) with a consideration of the ''budding'' and ''fission'' theories of twinning. Anat. Rec., vol. 92, pp. 1–16.



5 Heart and principal arteries of a 21-cm pig fetus, showing anomalous right subclavian artery attached to the pulmonary trunk. BT, bicarotid trunk. CC, costo-cervical. DA, ductus arteriosus. LCC, left common carotid. LIM, left internal mammary. LSC, left subclavian. LV, left ventricle. PA, pulmonary artery. RSC, right subclavian. RV, right ventricle. TC, thyro-cervical. V, vertebral. VN, vagus nerve.

A CASE OF ATYPICAL TWINNING IN FUNDULUS HETEROCLITUS

JANE M. OPPENHEIMER

Department of Biology, Bryn Mawr College, Pennsylvania and
Marine Biological Laboratory, Woods Hole, Massachusetts

ONE FIGURE

During the course of routine class experiments performed on eggs of Fundulus heteroclitus, a peculiar set of twin embryos was found which does not conform to the usual types of vertebrate twins. In the typical duplications described for teleosts, when two embryos are formed on a single yolk the longitudinal axes of the two components follow separate meridians of the egg, with the tails of the two individuals directed towards each other at early stages and the heads divergent. In the present case, the head of one embryo adjoined the tail of the other; the antero-posterior orientation of one of the embryos, therefore, was the reverse of the expected condition. Since the orientation of the two twin components with reference to each other raises an interesting problem relating to the dynamics of vertebrate gastrulation, it seems desirable to describe the embryos and to attempt an analysis of the possible mechanism by which they may have been produced.

The anomaly, which was called to my attention by Miss Liselotte Mezger of Bryn Mawr in the Embryology Course at Woods Hole, occurred in one of a batch of eggs treated at room temperature with a solution of $MgCl_2$ prepared by adding 22 cm^3 of molar $MgCl_2$ to 38 cm^3 of sea-water. The specimen had been placed in the solution 3 hours and 20 minutes after fertilization, remained in it approximately 45 hours, and was then transferred to sea-water.

The abnormality was first noted during the fourth day of development, at which time two embryos were found on the single yolk, one immediately anterior to the other, along approximately the same meridian of the egg.

During the fifth day, when the anomaly was first pointed out to the author, the more anteriorly located embryo had a fairly well-differentiated head, a short trunk and an abnormal tail (fig. 1). Its optic vesicles were grossly normal, as was the forebrain; the midbrain and hindbrain were slightly distorted. Two otic vesicles were present, both furnished with otoliths.

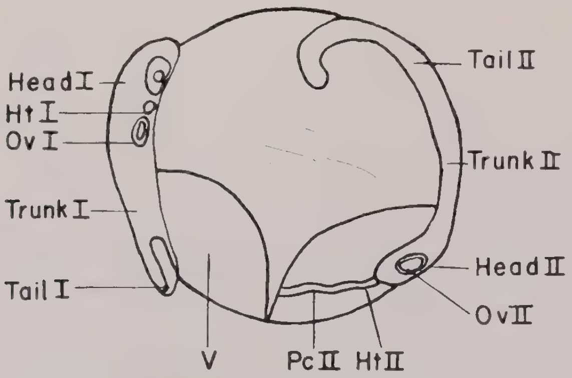


Fig. 1 Diagram illustrating position of twin embryos on yolk during fifth day of development. See text for description of embryos. Ov, otic vesicle; Ht, heart; Pc, pericardium; V, large blister-like vesicle in yolk.

A rhythmically contractile heart was present. The trunk, although abnormally short, exhibited contractility; a reduced irregular tail doubled back sharply to the right.

The second embryo, whose enlarged pericardium abutted upon the posterior region of junction of the anterior embryo and the yolk, approximated an anencephalic condition (fig. 1). At most a small amount of hindbrain was differentiated, and the single ear lacked an otolith. The trunk was reduced in width: the tail, however, was more nearly normal than was that of the anterior embryo, and exhibited vigorous lashing movements. The heart of the posteriorly located embryo was slender and threadlike, but was rhythmically contractile.

There was no circulation on the yolk-sac or within either embryo.

DISCUSSION

The development of the embryos of Fundulus heteroclitus, like that of other teleostean embryos, is markedly susceptible to modification by chemical treatment (cf. Stockard, '09, '10, '21), and twinning has frequently been reported as a result of such handling. The typical cases of twinning in teleosts are readily explained by the hypothesis that two centers of involution arise at separate regions on the edge of the blastoderm, each of these centers acting as an "organizer" determining the differentiation first of head and next of trunk structures as the blastoderm expands to cover the yolk. This concept is thoroughly in agreement with the established facts concerning the mechanics of gastrulation of vertebrates in general and teleosts in particular, and it explains satisfactorily the position and orientation of the longitudinal axes as they are found in the usually described types of twins.

The unusual orientation with respect to each other of the embryos described in this communication clearly negates the possibility that they have been formed in the usual manner of teleostean twins. It is evident that a duplication of the "organizer" in the usual sense cannot explain their origin.

One possible explanation of the formation of these twins might be that after the establishment of the embryonic shield, degeneration occurred in a band across the shield, effectively dividing the shield into an anterior and a posterior portion, each of which partially undergoes regulation to form an incomplete embryo. A certain amount of regulability has previously been demonstrated for the shield of Fundulus (Sumner, '03; Lewis, '12; Hoadley, '28; Oppenheimer, '36). Such an explanation, together with the orientation of the embryos, is compatible with the existence of an axial metabolic gradient as this has been postulated for the developing teleost egg by Hyman ('21) and Child ('43, '45).

Another explanation might be that involution occurred along a single axis of the blastoderm in two phases separated in time. This hypothesis would assume that gastrulation was initiated in the usual way, with the formation of a single embryonic shield, establishing the rudiments of the embryo described above as the more anterior of the pair of twins. It further assumes that for some reason the processes of involution were temporarily inhibited, in such a way that the overgrowth of the yolk by the blastoderm continued for a short period of time in the absence of the usual accompanying involution movements. Somewhat comparable overgrowth of the yolk by the "Deckschicht" of the blastoderm, after inhibition of involution movements, following experimental manipulation, has been described by Luther ('35) for the trout. The final assumption is that involution began again at a later time, following the original axis, these later processes of involution being responsible for the establishment of the posteriorly located embryo.

The fact that the head of the anteriormost embryo was more nearly normal than its tail, while the reverse condition characterized the posteriormost embryo, might be construed as supporting either hypothesis. In either case, it might have been expected that the posteriormost portion of the first embryo would have differentiated trunk rather than tail structures, and that the anteriormost portion of the second embryo would have differentiated trunk rather than head structures. The fact that the conditions were otherwise may be interpreted as corroboration of the ample evidence already at hand, namely, that the dorsal lip "organizer" is itself modifiable, regulating under experimental conditions in such a way as to produce as nearly complete an embryo as possible under whatever conditions it experiences, and that its regulation occurs in accordance with whatever laws exist to govern the production of an axial metabolic gradient in the vertebrate embryo.

SUMMARY

The treatment of an egg of Fundulus heteroclitus with a solution of $MgCl_2$ has resulted in the formation of a pair of twins whose orientation with reference to each other differs from that typical for vertebrate twins. One individual lay immediately anterior to the other, the longitudinal axes of the two following the same meridian of the egg, in such a way that the anterior structures of the posteriormost embryo adjoined the posterior portion of the anteriormost embryo.

As possible explanations of the origin of this anomaly, it has been suggested either that the shield was divided into two portions by a band of deteriorating cells, each portion undergoing some regulation, or that involution occurred along a single axis of the blastoderm in two phases separated in time. An overlap in the type of structures formed by the posterior end of the first embryo and the anterior part of the second is tentatively explained on the basis of the powers of regulation of the dorsal lip "organizer," and is compatible with the known facts concerning the existence of an antero-posterior gradient in the teleost embryo.

LITERATURE CITED

CHILD, C. M. 1943 Oxidation-reduction pattern in development of a teleost. Physiol. Zool., vol. 16, pp. 297–312.

——— 1945 A further study of oxidation-reduction pattern in teleost development. J. Exp. Zool., vol. 100, pp. 577–589.

HOADLEY, L. 1928 On the localization of developmental potencies in the embryo of Fundulus heteroclitus. J. Exp. Zool., vol. 52, pp. 7–44.

HYMAN, L. H. 1921 The metabolic gradients of vertebrate embryos. I. Teleost embryos. Biol. Bull., vol. 40, pp. 32–72.

LEWIS, W. H. 1912 Experiments on localization and regeneration in the embryonic shield and germ ring of a teleost fish (Fundulus heteroclitus). Anat. Rec., vol. 6, pp. 325–333.

LUTHER, W. 1935 Entwicklungsphysiologische Untersuchungen am Forellenkeim: Die Rolle des Organisationszentrum bei der Entstehung der Embryonalanlage. Biol. Zentralbl., vol. 55, pp. 114–137.

OPPENHEIMER, J. M. 1936 Transplantation experiments on developing teleosts (Fundulus and Perca). J. Exp. Zool., vol. 72, pp. 409–438.

STOCKARD, C. R. 1909 The development of artificially produced cyclopean fish — "The Magnesium Embryo." J. Exp. Zool., vol. 6, pp. 285–337.

——— 1910 The influence of alcohol and other anaesthetics on embryonic development. Am. J. Anat., vol. 10, pp. 369–392.

——— 1921 Developmental rate and structural expression: an experimental study of twins, "double monsters," and single deformities, and the interaction among embryonic organs during their origin and development. Am. J. Anat., vol. 28, pp. 115–277.

SUMNER, F. B. 1903 A study of early fish development. Experimental and morphological. Arch. f. Entw.-mech. d. Org., vol. 17, pp. 92–149.

BOOK REVIEW

BIOLOGICAL ACTIONS OF SEX HORMONES. By HAROLD BURROWS, C. B. E., Ph. D., F. R. C. S. The Macmillan Company, New York, x + 514 pp., 1945, $8.50.

This is essentially a detailed and co-ordinated digest of literature concerning the manner in which substances produced by the hypophysis, placenta, gonads and adrenals may influence the form and function of certain parts of the body. It deals almost entirely with biological experiments performed in the laboratory, many of them bearing on the relation of hormones to the formation of tumors. It is divided into six major parts: Gonadotrophins, Gonadal Hormones (in general), Androgens, Oestrogens, Progestins and Sex Hormones of the Adrenal Cortex. It contains 205 short tables of data but no photographs, an appendix of abbreviations, synonyms and proprietary preparations, a list of more than 1800 references to literature (titles being omitted), and a useful index comprising 61 pages.

The author presents an enormous amount of material from a vast literature on the subjects discussed; to do this so well without benefit of collaboration was truly a formidable undertaking. He places at our disposal many of those advances which have been made subsequent to publication of the second edition of Sex and Internal Secretions. Also, he brings to the fore considerable work of pioneers which takes on added significance when viewed in the light of recent observations. In presenting the results of experiments, he specifies the pertinent conditions under which they were obtained and he makes effective use of tabulations.

Embryologists in particular will be interested in his explanation of the hypospadias that may be induced experimentally by subjecting mammalian embryos to the influence of certain steroid hormones. He says that these hormones act by causing the uniting edges of the urethral folds to become so differentiated ("keratinized") that union cannot be completed. He supports this view by citing observations on such vital phenomena as the healing of wounds, the amalgamation of malignant papillomata and the separation of the prepuce from the glans penis.

Although it would seem that coverage of the British literature on the subjects under consideration is complete indeed, there are many instances in which topics are not brought up to date — doubtless due largely to the difficulty of obtaining foreign periodicals during the war. Thus, regarding bleeding from endometrial grafts, the only reference of Markee's work is based upon an abstract dated 1933. Concerning the effects of androgen upon spermatogenesis, nothing is said about experiments in which crystalline androgen was inserted into the substance of the testis. In discussing the effects of hormones upon the developing organs of the opossum the author seems to have missed the significant contributions of Carl Moore. Other subjects which one would like to have had discussed include the "carmine cell" of the hypophysis, the effects of the adrenal cortex upon lymphoid tissue and the series of papers on relaxin which appeared in 1944.

There are places, also, in which the usual precision of the author is not sustained, as in the reference to estrous cycles in women (pp. 393 and 410) and monkeys (p. 411) and in the statement that "normal female rats and mice possess a penis somewhat like that of the male" (p. 289). Concerning Burns' observation ('39) that in male opossums receiving androgen the testes fail to descend, the author misconstrues this to mean that the androgen acted by interfering with the supply of hypophyseal gonadotrophin (p. 205); actually, however, as clearly stated by Burns, it was the anomalous development of a uterus which prevented the testes from descending. Equally ineffective is the citing of Tschopp's paper in support of the view that in castrated male rats injections of estrogen cause the bulbo-urethral glands to exhibit secretory activity, for this article does not deal with the minute anatomy of the bulbo-urethral glands (p. 112).

With respect to the order of topics, one wonders why the author presents the effects of estrogen upon the accessory reproductive organs of the male before taking up the effects of this hormone upon the various organs of the female. Similarly, he discusses the effects of androgen upon ovulation before recording the effects of it upon spermatogenesis and the action of progesterone on the seminal vesicles before noting its action on the uterus.

Typographical errors are relatively scarce, the following being examples: ovine (p. 77), prophylene (p. 129), Overholser and Warren (for Overholser and Nelson, p. 115), Moore, Lamar and Back (for Moore, Lamar and Beck, p. 129) and p. 277 (an erroneous cross reference which appears on p. 269).

In conclusion, this book is a distinct contribution to the physiology of reproduction and to the biology of tumors. It will be quite useful to those investigators who are pursuing problems in these and related fields, and should constitute a fertile source of ideas for graduate students who are exploring various subjects with a view to selecting problems for research.

L. J. Wells
University of Minnesota

BOOK REVIEW

PHYSICAL CHEMISTRY OF CELLS AND TISSUES. By RUDOLF HÖBER with the collaboration of David I. Hitchcock, J. Bateman, David R. Goddard, and Wallace O. Fenn. pp. 1–676. The Blakiston Company, Philadelphia, and Toronto.

Since the publication of the first edition of his Physikalische Chemie der Zelle und der Gewebe in 1902, Professor Höber has accepted the task of interpreting the progress of physical chemistry for the benefit of biologists. This work in its several editions has held for many years an honored place on the work-table of biologists. The new work, while different in important respects from its German predecessors, is also a valuable contribution to current research. Professor Höber has perceived the growing importance to biological research of the recent advances in molecular physics and the introductory chapters by Davidson and Bateman are devoted to this theme. These chapters will prove to be of inestimable value to those students of the fundamental properties of living substances and of life processes who have sufficient knowledge of physics, chemistry and mathematics to comprehend them. The reader must remember, however, that while these discussions will aid him to interpret the results of his investigations, they do not allow him to predict them. Even scientists often prefer argument to work.

Bateman's discussions of condensed systems of large molecules and of certain properties of films and membranes are particularly apposite to the problems of protoplasmic structure. One is entitled to doubt, however, whether the physicist who derives certain laws of intermolecular relations by the study of simplified mimetic systems fully appreciates the degree of complexity present in the molecular aggregates of protoplasm. When he does, he may come to regard such a diagram of the plasma membrane as figure 27, page 274, as a gross caricature.

The functional and structural applications of physical principles, with the exception of the excellent chapters on respiration and on muscle by Goddard and Fenn are undertaken by Professor Höber himself. These chapters are marked by the same profound knowledge of the literature as has characterized the earlier editions in German.

One could wish that, for the sake of historical perspective, the important contributions of Nägeli, Pfeffer, and De Vries had been more fully abstracted, and that much pertinent material that has appeared in anatomical journals had not been overlooked. On the other hand, Professor Höber's chapters are strictly objective and free from the flavor of propaganda which mars so much scientific writing.

This reviewer does not share Professor Höber's enthusiasm for the results of perfusion experiments in the frog's kidney which he regards as unphysiologic. He also notes with regret that the details of the fundamental experiment of Nussbaum on the frog's kidney has at last been wholly eliminated. This experiment has not lost its validity in spite of the many abortive attempts to repeat it by experimenters unfamiliar with the vascular anatomy of the frog.

The discussion of the secretion of the gastric juice is marred by the apparent lack of adequate knowledge of the structure of the gastric gland and the results of histologic investigations on this topic. The fact that the portion of the gastric gland in which the parietal cells are most abundant is also occupied by a special type of cell peculiar to that region, a fact known to histologists for half a century, has been wholly ignored.

The casual reader will be disappointed to discover that after more than 50 years of research, we still do not know the composition or the thickness of the plasma membrane, though much has been learned about its permeability and the factors which influence it. This reviewer may be permitted to express the hope that a better understanding of the conditions of aggregation of those molecular species which are actually present in protoplasm as revealed by such experiments as those of De Jong on coacervation and the study of the microscopic and submicroscopic aggregates of protoplasm itself may soon throw much light on this problem.

Professor Höber and his collaborators and the Blakiston Company are to be congratulated on the production of a book which will serve to broaden the training of biologists and also save them much weary search of the literature.

R. R. Bensley
University of Chicago

BOOK REVIEW

AN INTRODUCTION TO PHYSICAL ANTHROPOLOGY. By M. F. Ashley Montagu. Springfield, Illinois, Charles C. Thomas, 1945. Pp. xiv and 325. $4.00.

Those who wish a survey of the recent findings in physical anthropology will be grateful for this book. The format is attractive, the content is interesting and enlightening. There is a classification of the Primates and a description of their distinguishing traits. There are sections devoted to, respectively, the origin and evolution of man, the divisions and ethnic groups of man, heredity and the influence of environment, and a concluding section describing the fundamental anthropometric techniques.

The discussion of the relation between race type and cultural traits is incisive and convincing.

When Dr. Montagu defined the affix platy as ''down-pointing,'' (12), he had certainly not consulted his Greek dictionary. Nor can I follow him when he asserts the Darwin doctrine (62) that ''man originated from an early anthropoid ape'' as ''so fully substantiated.'' For all the evidence to the contrary, that early common ancestor was as much man as anthropoid ape.

Also, the reviewer must wonder why Darwin's The Descent of Man should be considered ''the classic work on the probable manner of origin and evolution of man'' (62). Except for alleging a close relationship between man and anthropoid ape, there is little in that book which present day or recent anthropology accepts. Further, F. Wood Jones can scarcely be said with accuracy or meaning to hold that ''man is derived from an anthropoid stock'' (63). Anthropoid means manlike; and Wood Jones does not derive man from an anthropoid ape stock. On page 69 we meet with the old assertion that the endocranial surface of the Pithecanthropus erectus skull indicates that he ''may have used some form of language.'' Thirty years ago Symington showed, clinically, that there is little relation between the position of the convolutions of the brain surface and the markings on the endocranial vault. If by examining a skull — or a brain — of present day man one can not tell whether the individual was a mute or an orator, how can one make a pronouncement about the speech ability of the carrier of a skull millennia ago — a skull which some regard as gibbonoid?

The "more than fourteen individuals" of the Sinanthropus pekinensis type (73) should, according to Weidenreich, be "at least forty." On page 106 we meet with another old stereotype, namely, that the increase in the convolutions of the brain is responsible for mental development. The degree of convolution in man, relative to cubic content, decreases with growth of the brain, in the individual; and some of the anthropoid apes have in most portions of the brain a relatively more convoluted surface than has man. Again, we are told (110) that some of the early preEuropean migrations into the New World were "almost certainly of Caucasoid type." The evidence for this I do not know; other than variability in the Indian type. But it has not been shown that such variability is more than normal. And whence these presumed Caucasoid types came, if they came from Asia, is a puzzle.

The suggestion (147–148) that sexual selection within a primitive group gave rise to distinct types seems of doubtful value. In any preliterate group known to us everybody marries; and unless we suppose a differential fertility linked with type of hair, or color of eye, or other physical trait, it is difficult to see how sexual selection could have modified the physical type of the group.

Then, too, those "dangerous ultra-violet rays of the tropical sun." They are not nearly as dangerous in the tropics as in the arctic regions. The black skin should have shown its survival value in Scandinavia rather than in the humid moisture-laden lands of the equatorial belt of Africa.

True Negroes are said to be "relatively short-legged"; whereas a notable difference between Caucasoids and Negroids of the same stature is the greater length of legs in Negroes. To classify Australians and Ainu as Caucasoids seems to be a measure of despair (171); and to allege "a striking resemblance" between these first two groups is stretching the tenuous beyond the breaking point. What these two groups have in common besides a heavy pilosity I do not know; and the Australian skull seems about as far removed from the average Caucasoid skull as anything we could find this side of Neanderthal man.

Nor do I see any reason to classify Bushmen-Hottentot as a "strong Mongoloid admixture" (164), just because these Negroids have a sacral spot in infancy and a yellowish tinge in the pigment. How in the name of all that is probable could they have acquired that admixture?

There is no evidence that the Ainu ever inhabited the Riukiu Islands (193). In historic times they have not been further south than central or northern Honshu.

An examination of the table on page 241 which indicates changes in cephalic index and in stature of children of immigrants (Boas' data) shows that with one exception, in the eight groups, an increase in stature is accompanied by a decrease in cephalic index, and a decrease in stature is accompanied by an increase in cephalic index. This negative correlation is the usual correlation, both during growth, and for adults.

The changes in cephalic index, therefore, may be considered at least in part a function of changes in stature — and the data, so far as they have been analyzed and presented, do not indicate more than this. Moreover, the native American population of American parentage has been changing, at least during the years of growth; and a similar phenomenon has appeared in Sweden and elsewhere.

Wilson D. Wallis
University of Minnesota

IS THE CONCEPT OF THE "ANATOMICAL POSITION" STILL TENABLE? A CONTRIBUTION TO THE SUBJECT OF ANATOMICAL NOMENCLATURE

ERNEST LACHMAN

Department of Anatomy, University of Oklahoma School of Medicine, Oklahoma City

It is not surprising that our anatomical terminology reflects man's greatest biological achievement, the upright position. Such terms as "superior" and "inferior," "ascending" and "descending," "anterior" and "posterior," which are retained in our modern anatomical vocabulary, are meaningful only as applied to the erect attitude. The latter is commonly referred to as the "anatomical position" (Cunningham, '43). "The cadaver may be placed on the table lying on its back, on its side, or on its face, but for descriptive purposes it is assumed to be standing erect in the Anatomical Position" (Grant, '44).

The employment of these terms and of the underlying positional concept can be traced back to medieval anatomy, as exemplified by the use of the term "aorta descendens" as early as 1523 by Berengar of Carpi (Singer, '25). It is further illustrated by the classical drawings in Vesalius' Fabrica, which picture the cadaver in the upright position. In this connection it is interesting to note that early medieval anatomists undertook some of their dissections with the body suspended vertically (Singer, '25). Very few attempts have been made in modern times to follow this example although it might have furnished fruitful information. Thus, Mueller ('23) studied the topographic anatomy of the viscera of freshly executed individuals whose bodies had been embalmed in the upright position.

THE ANATOMICAL RECORD, VOL. 95, NO. 2
JUNE, 1946

Frequent objections have been voiced to a terminology which is based on the vertical position. So far they have come mainly from two sources: the comparative anatomist and the teaching clinician. Since the erect attitude has been made the basis for description of the various structures and their relationship, the terminology used in human anatomy is often not applicable in zoology and comparative anatomy. "The lack of correspondence between the actual position of the chief axis of man and the horizontal fore- and aft-axis of vertebrates in general, results in discrepancies when the three principal planes of the human body are compared with those of other animals" (Piersol, '36). H. Richter ('28) calls attention to the fact that the plane which divides the body into ventral and dorsal halves is designated as "frontal" in man and as "horizontal" in animals which may, of course, lead to confusion. A unified terminology which disregards man's peculiar posture would be very desirable from the point of view of comparative anatomy. Embryology which represents a connecting link between comparative and gross anatomy cannot easily employ such misleading terms as "anterior" and "posterior," "superior" and "inferior," since the fetus in utero does not accommodate itself to the "anatomical position" for the convenience of embryologists. Thus we speak of ventral and dorsal mesenteries and of ventral and dorsal derivatives of the musculature instead of using the confusing terms "anterior" and "posterior." We also employ the terms "cranial" and "caudal" rather than "superior" and "inferior."

Of greater practical importance are the objections to our terminology raised by clinicians, particularly those in the teaching field. A good example are the statements by Sellheim ('27), an outstanding teacher of obstetrics. He calls attention to the terminological difficulties that may arise in the examination of pregnant and parturient women. Directional terms that are derived from the upright position can be applied to the horizontal posture of the patient intra partum only by a complicated mental process of conversion that may lead to ambiguities and serious misunderstandings. Exception has been taken from the surgical side to such terms as ''upper'' and ''lower'' end of a bone, since the upper end may be at a lower level than the lower end, as e.g., in the upraised arm. Again this criticism can be easily overcome by the employment of terms that are applicable irrespective of the position of the body.

Recently these objections to positional terms, which are predicated on the erect posture, have been restated and summarized by the Stanford group of anatomists (Greulich, etc., '45) in connection with a reconsideration of the whole problem of anatomical nomenclature. Thus the time is ripe for a fresh evaluation of the concept of the ''anatomical position.'' It is the purpose of this paper to point out important contradictions which pervade our teaching of surface and relational anatomy and which can be attributed to the retention of this concept.

If according to Cunningham ('43) ''all topographical descriptions are framed on the anatomical convention that the body is in the erect position,'' then radiologic investigation of visceral topography in the upright living should furnish results which harmonize with the findings of conventional anatomy. The roentgen-anatomical literature of recent years gives proof to the fact that the deductions arrived at by the two methods often do not conform at all.

The subject of abdominal topography serves as a good example of the problem in question. Textbook presentations on relationship and surface anatomy of the abdominal viscera are essentially based on studies of cadavers which were undertaken by the English Anatomist, Addison, at the turn of the century (1899–1901). As his figures demonstrate (Morris, '42), Addison divides the anterior surface area of the abdomen in small squares and designates to each organ its place in this scheme based on its average position in the embalmed cadaver. Thus the pylorus is assigned a position on the transpyloric line which goes through the disc between the first and second lumbar vertebra. In contrast to Addison's findings, x-ray investigations in the living have shown that "the normal abdominal viscera have no fixed shapes and no fixed positions," and that "every description of them must be qualified by a statement of the conditions existing at the time of observation" (Barclay, '36). Thus, Moody and his co-workers ('29) in their x-ray studies on healthy young adults in the erect, i.e., "anatomical position" showed that "the pylorus was as high as the transpyloric plane in none of the males and in only 3.4% of the females." Similar discrepancies between conventional presentations on topographic anatomy and the results of x-ray investigations are found for the duodenum, gall-bladder, coecum, transverse colon, spleen, liver and kidney, particularly if the anatomical position is used as the basis for topographic definitions. It is evident that the divergencies are not due to differences in the method of study, dissection on the one hand and radiography on the other, but to a difference in the objects explored, namely, the recumbent cadaver in the former instance and the living subject in its varied posture in the latter.

With respect to the thoracic viscera the belief is usually held that their position is not subject to marked variations. Again, as in the abdomen, conditions found in the supine cadaver are presented as the norm also for clinical purposes. Little or no attention is paid to temporary physiologic alterations in the site and configuration of the thoracic organs and to constitutional variations from individual to individual. Thus the bifurcation of the trachea is usually placed at the level of the intervertebral disc between fourth and fifth thoracic vertebra, while roentgenography reveals that in the upright adult subject it is usually considerably lower (Koehler, '35; Kreuzfuchs, '37).

X-ray studies on the surface projection of the heart in the erect subject in the midphase of respiration have shown that the organ in this position lies considerably lower than is usually assumed (Mainland and Gordon, '41). Particularly surprising is the location of the inferior cardiac border which is given as crossing the midline on the average 4½ cm below the xiphisternal joint (Mainland, '45). Although the position of the heart valves cannot be determined directly on the x-ray film and sufficient studies with contrast media in the normal living individual are not yet available, it can be deduced from the data at hand that the standard anatomical diagram of the surface projection of the cardiac valves is not correct for the upright individual.

Results of roentgenographic studies on posterior mediastinal and posterior inferior boundaries of lung and pleura in the erect subject are likewise not in complete agreement with conventional anatomical teaching. According to the latter the lungs and pleural cavities are separated posteriorly by a wide mediastinal space in which the oesophagus is located directly in front of the spinal column. In contrast to this roentgenograms occasionally depict the presence of a prevertebral space which is occupied by lung and pleura (Danelius, '31; Lachman, '42). It cannot be ascertained at the present time how common this finding is. Figures given in the literature range from 30 to 70% (Stéphani and Kirsch, '36; Heiss, '19). The location of the posterior inferior extent of lung and pleura in the upright individual differs likewise from standard descriptions. It can be determined rather easily by radiography and is commonly found one and occasionally two vertebrae lower than given in anatomical presentations. The configuration of the pleural reflection at this site is also at variance since it often shows a previously undescribed curvature (Lachman, '42).

Enough data have been presented to prove that the concept of the upright posture as "the anatomical position" is not consistent with conventional topographic descriptions since these are based on studies of supine cadavers. Actually the statement in Cunningham ('43) that "all topographical descriptions are framed on the anatomical convention that the body is in the erect position" does not mean what it implies, but refers to the erect posture as the basis of orientation for terminological purposes only. The variations in the location of the viscera due to positional changes were not known when the concept of the "anatomical position" was introduced.

But if this concept is to be retained, then complete revision of positional and relational anatomy based on studies in the erect living subject is necessary. So far only insufficient radiographic data on shape, relation, and position of the viscera are available for this purpose. Thus for the time being it seems more advisable to discard the concept of the "anatomical position" altogether and replace directional and positional terms, which are predicated on the erect posture, by designations that are independent of the position of the body.

LITERATURE CITED

ADDISON, C. 1899–1901 On the topographical anatomy of the abdominal viscera in man, especially the gastro-intestinal canal. J. Anat. and Physiol., vol. 33, pp. 565–586; vol. 34, pp. 427–450; vol. 35, pp. 166–204.

BARCLAY, A. E. 1936 The digestive tract. A radiological study of its anatomy, physiology and pathology. Cambridge.

CUNNINGHAM, D. J. 1943 Textbook of Anatomy. 8th ed. New York.

DANELIUS, G. 1931 Roentgenologie der oberen medialen Lungenabschnitte. Fortschr. a. d. Geb. d. Roentgenstr., vol. 44, pp. 626–634.

GRANT, J. C. B. 1944 A method of anatomy. 3rd ed. Baltimore.

GREULICH, W. W. ETC. 1945 Terms of position and direction in the NK-INA Revision of the Basle Nomina Anatomica. Anat. Rec., vol. 92, pp. 359–362.

HEISS, R. 1919 Ueber die hinteren Pleuragrenzen. Arch. f. Anat. u. Physiol., Anat. Abt., pp. 130–136.

KOEHLER, A. 1935 Roentgenology. 2nd ed. Baltimore.

KREUZFUCHS, S. 1937 Die Bifurkation der Trachea in Konstitution und Pathologie. Fortschr. a. d. Geb. d. Roentgenstr., vol., 56, Beiheft 2, pp. 22–24.

LACHMAN, E. 1942 A comparison of the posterior boundaries of lungs and pleura as demonstrated on the cadaver and on the roentgenogram of the living. Anat. Rec., vol. 83, pp. 521–542.

MAINLAND, D. 1945 Anatomy as a Basis for Medical and Dental Practice New York.

MAINLAND, D., AND E. J. GORDON 1941 The position of organs determined from thoracic radiographs of young adult males, with a study of the cardiac apex beat. Am. J. Anat., vol. 68, pp. 457–496.

MOODY, R. O., R. G. VAN NUYS AND C. H. KIDDER 1929 The form and position of the empty stomach in healthy young adults as shown in roentgenograms. Anat. Rec., vol. 43, pp. 359–379.

MORRIS 1942 Human Anatomy. 10th ed. Philadelphia.

MUELLER, F. W. 1923 Untersuchungen ueber die Topographie der Rumpfeingeweide bei verschiedenen Stellungen des Koerpers. Zeitschr. f. d. ges. Anat., 1. Abt., vol. 67, pp. 1–190.

PIERSOL, G. A. 1930 Human Anatomy. 9th ed. Philadelphia.

RICHTER, H. 1928 Zur Nomenklatur der Koerperrichtungen bei Tier und Mensch. Anat. Anzeiger, vol. 66, pp. 263–264.

SELLHEIM, H. 1927 Die normale Geburt. In J. Halban and L. Seitz: Biologie und Pathologie des Weibes, vol. 7, pt. 1, pp. 1–307. Berlin.

SINGER, C. 1925 The evolution of anatomy. London.

STÉPHANI, J., AND R. KIRSCH 1936 Étude radiographique de l'expansion interne du poumon droit. Condition nécessaire pour qu'elle se réalise et que son image radiographique apparaise. Press. méd., vol. 44, pp. 1585–1586.

IN VIVO STAINING AND RETARDATION OF GROWTH OF SARCOMATA IN MICE[1]

MARGARET R. LEWIS, HENRY A. SLOVITER AND PHILIP P. GOLAND

The Department of Embryology of the Carnegie Institution of Washington, Baltimore, Maryland, and The Wistar Institute of Anatomy and Biology; The Harrison Department of Surgical Research and the Department of Neurosurgery, School of Medicine, the University of Pennsylvania, Philadelphia.

INTRODUCTION

In an effort to determine why certain forms of penicillin destroyed malignant cells growing in tissue cultures but not in vivo (Cornman, '44 and Lewis, '44) studies were made of the tumor-inhibiting value of various pigments extracted from molds and other plants. As a part of this study a number of coal-tar dyestuffs were also evaluated. The excretion of certain orally administered substances without intra-vitam staining of the animals tissue led us to repeat earlier investigations on vital staining of normal and malignant cells. As a result of these studies it was found that sarcoma cells became selectively stained in mice fed with Nile blue, but not in those fed with other dyes. In no instance was staining of tumor cells observed in mice which received dyes parenterally.

It therefore, seemed of moment to study all available forms of Nile blue as well as dyes of the same (oxazine) and other series, especially those closely related in chemical structure such as the thiazine dyes.

MATERIAL AND METHOD

Through the kindness of manufacturers[2] twenty-five samples of Nile blue, samples of forty-two dyes of the same

---

[1] Aided by a grant made to Dr. Warren H. Lewis from the International Cancer Research Foundation.

[2] A list of the sources and identity of all these dyes will be reported later.

(oxazine) group and samples of thirty-one dyes of other series were obtained and tested.

It was found that different samples of commercial Nile blue varied considerably in their degree of tumor-inhibition and toxicity and that even samples of different batches from the same manufacturer were not uniform in action. Accordingly, samples of pure Nile blue were synthesized and tested. Thorpe ('07) showed that Nile blue is readily converted to the corresponding oxazone and that the conversion explains the behavior of solutions of Nile blue when used for histological staining. In view of this, oxazones were prepared for this investigation from different samples of Nile blue and pure synthetic oxazone was also prepared and tested.

Mice 30 to 40 days old, weighing 18 to 20 gm, were implanted with a graft (4×2×1 mm) of a transplanted sarcoma that originated in a mouse of the same inbred strain. The mice were of the inbred Bagg albino and of the inbred black agouti strains which have been inbred in our laboratory for the past 10 years. During that time many thousands of these mice have been ingrafted with a tumor from a mouse of its own strain and every one of the grafts grew into a large tumor; not one regressed.

Each mouse was kept separately in a well ventilated tin box containing shavings and supplied with a water bottle and food cup. The desired amount of each dye was ground in a mortar together with 100 gm of pulverized Purina fox chow. The mixture was stored in a closed light-resistant glass jar.[3] This was sufficient for one mouse for about 10 days. An abundant quantity of the mixture of dye and food was placed in the food cups each day. Owing to the fluorescent nature of some of the dyes, bright light was excluded from the animals except for a short period of time during their daily examination.

In some experiments many percentages of dye were tested (0.15, 0.2, 0.25, 0.3, 0.35, 0.4, 0.45 and 0.5%); in others only 2 percentages (0.25 and 0.5%) were used while the majority of the dyes were tested in 0.4% concentrations. Each series of tests were repeated one or more times.

---

[3] We are indebted to Mrs. Wm. T. Higgens for the special glass jars.

In some experiments treated mice were permitted to live as long as possible after the controls died, but in the majority of tests the mice of a series were sacrificed and autopsied when the controls died or had large tumors (about 30×20×18 mm). If a dye was so toxic it brought about the early death of the mouse to which it was administered, the test was repeated using smaller amounts of dye until a non-toxic percentage was found.

Specimens of urine from the treated mice were collected on white filter paper every few days beginning 17 to 20 hours after the dye-containing food was made available. The size of the tumors were recorded at intervals of several days.

Owing to the concentration of bile in the gall bladder and of urine in the kidney (Rous and McMasters, '19; Richards, '29 and Marshall, '34) the presence of a dye and its approximate concentration in the circulation of an animal could be estimated by an examination of its bile and urine. In our experiments we found this a useful method of determining whether a dye given orally was present in the circulation and so carried to the animal's tumor tissue. Certain colorless intermediates used in synthesizing dyes could not be detected in this way; fortunately chemical tests of urine could be made for these substances. Such tests showed that alpha naphthol, diethylaminophenol and nitrosodiethylaminophenol were present in the circulation of mice to which they had been fed.

RESULTS

Every sample of Nile blue and its oxazone fed to mice stained their tumors a diffuse blue and retarded their growth while none of the other dyes either stained tumors or retarded their growth. Oxazone fed to mice stained their tumors blue, the same shade as that of tumors in mice fed Nile blue, although oxazone is a fat soluble red dye.

The diffuse blue stain was not restricted to the tumor alone, but appeared to a slight extent in the lymph nodes, thymus and testicles of mice whose tumors were stained. The depth of staining that occurred in these organs depended largely upon the kind of Nile blue or its oxazone used. A few Kupffer cells in the liver, spleen, omentum and medulla of the adrenal contained blue stained masses, apparently ingested damaged blood cells that had become stained.

All the Nile blue dyes were excreted in the urine and bile. The depth of color in these fluids depended upon the amount and kind of Nile blue fed to the mouse. It graded from blue lavender to dark purple. Oxazones were excreted in the urine and bile as pink, red or plum colored fluid matter depending upon the oxazone fed. The majority of oxazone dyes and many dyes belonging to other groups were excreted in the bile and urine, and, therefore, were undoubtedly present in the circulation. From this we may conclude that the failure of tumor tissue to become stained in mice fed with other dyes was not due primarily to lack of contact of the cells with the dyes.

Typical vital staining such as that described with other dyes was difficult to obtain with Nile blue even when the dye was injected owing to its precipitation in salt solution and in body fluids. However, if the dye was dissolved in 5% glucose in water it was not so readily precipitated and when glucose solutions of Nile blue were used parenterally or supra vitally the usual type of vital staining took place. Intra vitam staining of granules, vacuoles and specific bodies did not occur in tissues of mice fed with any dye. Nile blue is listed as a fat stain, but fat globules were not stained in living tissue. In order to demonstrate the simultaneous staining of neutral fat and fatty acid as described by Smith ('08) it was necessary to fix the tissue in formalin before staining (Lewis, '18, and Lewis and Lewis, '15). Certain other fat stains, Sudan III and scarlet red, failed to stain fat globules in mice to which they were fed except in those instances in which they were dissolved in oil as recommended by Riddle ('10). Oxazone which is fat soluble failed to stain fat.

The growth of the tumor grafts was retarded in all mice fed Nile blue. The amount of retardation depended upon the kind and amount of Nile blue fed to the host. Some Nile blue samples were more toxic than others. These were fed in 0.1% instead of 0.4% mixtures with the food. At the time of autopsy some saracomas were about 1/10, others about 1/20, the size of those that had grown for the same length of time in mice fed pulverized food without dye. Sarcomas in mice fed with certain kinds of Nile blue were so small that only a rim of blue tissue was present around the dead grafts. Microscopical preparations of even the smallest tumors showed viable tumor cells, some in mitotic division, in the blue stained portion of the tumor while the unstained portion was made up of degenerated and fragmented tumor cells. When the blue portion of small and of medium sized tumors was transplanted the grafts from the small tumors grew much more slowly than those from the medium sized tumors. Samples of different batches of Nile blue from the same manufacturer exhibited differences in their tumor-inhibiting factor, but not necessarily in the depth of tumor staining. The oxazone likewise retarded growth of sarcomas, but the degree of retardation varied among the various samples used.

DISCUSSION

Many studies by other investigators have been made on intra-vitam staining in which more than 150 dyes were used. Dyes were administered orally in only five of these investigations. Hashida ('41) and Amano and Hashida ('41) compared the effect of feeding thionin, toluidin blue and Nile blue sulphate together with the cancer producing o-amidoazotuluol on the development of hepatomas in rats. They found that the addition of these dyes to this carcinogenic agent reduced the number of hepatomas that eventually developed in the treated animals. Rats fed with Nile blue sulphate developed less than half as many (20%) hepatomas as those fed with thionin or with toludin blue (50%) while every rat that received the carcinogenic agent without one of these dyes developed a hepatoma. Ito ('41) claimed that methylene blue had a cancer inhibiting action when fed together with o-amido-azotuluol. When the methylene blue was given after malignant cells had begun to develop, about 250 days, the number of hepatomas was not decreased. Jacobi ('06) treated patients with inoperable cancer by means of pills containing methylene blue. He claimed that the treatment prolonged their lives although it failed to cure the disease. Copeman, Coke and Gouldesbrough ('29) gave fluorescein by mouth in order to color lesions that were to be irradiated. These observers were not interested in vital staining. Hashida, Amano and Ito did not mention the excretion of the dyes or whether the cancer tissue became stained. Jacobi noted that methylene blue was excreted by the kidney, and Copeman, Coke and Gouldesbrough mentioned that the dye was excreted in urine and that the tumor tissue was not stained.

Other investigators, Evans, Ludford, Simpson, Moribe, Kiyono and Zahl) who studied dyes as therapeutic agents or for vital staining of tissues administered them by intratumor, subcutaneous, intravenous or intraperitoneal injection. Probably the reason for this is due to the fact that when dyes are given by mouth the type of staining known as vital staining does not occur. Vital staining results when living cells are brought into contact with the dye directly either in tissue cultures, thin spreads or by injection into an animal. This results in staining of preformed bodies such as mitochondria, granules, vacuoles, also of phagocytosed particles and pinocytosed fluids located in the uncolored cytoplasm of living cells.

In our investigations we used all the classical vital dyes such as trypan blue, isamine blue, methylene blue, brilliant cresyl blue, congo red, fuchsin, safranine and many others, both in vitro and in vivo. We found that these dyes brought about vital staining in animals when injected but not when given orally. When dyes are taken up from the digestive tract vital staining did not occur even though, judging from the color of the host's urine, dyes were present in the circulation.

The diffuse staining that occurred in mice fed with Nile blue or its oxazone did not resemble what is known as vital staining, since colored bodies were not present in cells otherwise unstained; instead the whole cell was diffusely colored. Even in microscopical preparations it was impossible to determine the site of the staining owing to the lack of contrast in the stained tissue.

SUMMARY

The effect of 114 dyes administered parenterally and orally to tumor ingrafted mice was studied. It was found that intra vitam staining, better known as vital staining, occurred in mice that received certain dyes parenterally, but not in those fed with the same dyes even though in both experiments dyes were excreted in the urine and bile of the treated animals.

In mice injected with the majority of vital dyes the capsule but not the inner viable portion of their tumors became vitally stained. In contrast the tumor tissue became diffusely stained. while its capsule and surrounding tissue remained unstained in the mice fed with Nile blue or its oxazone. The diffuse blue staining that took place in sarcomas of mice was quite unlike vital staining. None of the other dyes tested stained sarcoma cells regardless of whether injected or given by mouth.

Marked retardation of growth of sarcomata in mice was obtained by feeding Nile blue. The oxazone likewise retarded growth of tumors. The degree of retardation varied among the various samples used.

CONCLUSION

Diffuse staining of the viable portion of sarcomata in mice was achieved by feeding Nile blue or its oxazone. Simultaneous retardation of growth of the sarcomata took place. Neither of these effects was observed with any other dye.

LITERATURE CITED

Amano, S., and M. Hashida 1941 Kanzerogener sowie antikanzerogener Farbstoff. I. Mitt. Gann, vol. 35, p. 296.

Cornman, W. I. 1944 The survival of normal cells in penicillin solutions lethal to malignant cells. Science, vol. 99, p. 247.

COPEMAN, S. M., F. COKE AND C. GOULDESBROUGH 1929 "Activated" (irradiated) fluorescein in the treatment of cancer. Brit. Med. J., vol. 2, p. 233.

DURAN-REYNALS, F. 1939 Studies on the localization of dyes and foreign proteins in normal and malignant tissues. Am. J. Cancer, vol. 35, p. 98.

EVANS, H. M., AND K. J. SCOTT 1921 On the differential reaction to vital dyes exhibited by the two great groups of connective-tissue cells. Cont. Embry, vol. 10, no. 47.

HASHIDA, M. 1941 Über die antikanzerogenität von künstlichen anilinfarbstoffen bei der experimentellen hepatomerzeugung I. Mitt. Gann, vol. 35, p. 164.

ITO, S. 1941 Über die hemmende Wirkung von Methylenblau bei experimenteller Hepatomentstehung. II. Mitteilung, Gann, vol. 35, p. 182 and p. 346.

JACOBI, A. 1906 Methylthionin hydrochlorid in inoperable cancer. J. Am. Med. Assn., vol 47, p. 1545.

KIYONO, K. 1914 Die vitale Karminspecherung. Gustav Fischer, Jena.

KISIMOTO, S. 1940 Experimental study of effect of aniline dye upon the growth and radiosensibility of transplanted malignant tumor. Jap. J. Obstet. and Gyn., vol. 23, p. 68.

LEWIS, M. R. 1918 The formation of the fat droplets in the cells of tissue cultures. Science, vol. 48, p. 398.

LEWIS, M. R. 1944 The failure of purified penicillin to retard the growth of grafts of sarcoma in mice. Science, vol. 100, p. 314.

LEWIS, M. R., AND W. H. LEWIS 1915 Mitochondria (and other cytoplasmic structures) in tissue cultures. Am. J. Anat., vol. 17, p. 339.

LUDFORD, R. J. 1929 The vital staining of normal and malignant cells. II. The staining of malignant tumors with trypan blue. Proc. of Royal Soc. of London, Ser. B, vol. 104, p. 493.

MARSH, M. C., AND B. T. SIMPSON 1927 Chemotherapeutic attempts with coal tar derivatives on spontaneous mouse tumors. J. Cancer Research, vol. 11, p. 417.

MARSHALL, E. K. 1934 The comparative physiology of the kidney in relation to theories of renal secretion. Physiol. Rev., vol. 14, p. 133.

MORIBE, R. 1932 Studies on the vital staining with oxazin-, thiazin- and azin group. Trans. Jap. Path. Soc., vol. 22, p. 462.

RICHARDS, A. N. 1929 Methods and results of direct investigation of the function of the kidney. Williams and Wilkins, Baltimore, Md.

SMITH, J. L. 1907 On the simultaneous staining of neutral fat and fatty acid by oxazine dyes. J. Path. and Bact., vol. 12, p. 1.

THORPE, J. F. 1907 A reaction of certain coloring matters of the oxazine series. J. Chem. Soc., vol. 91, p. 324.

ZAHL, P. A., AND L. L. WATERS 1941 Localization of colloidal dyes in animal tumors. Proc. Soc. Exp. Biol. and Med. vol. 48, p. 304.

FURTHER INVESTIGATIONS ON THE EFFECT OF THE MALE SEX HORMONE ON ENDOCHONDRAL OSSIFICATION

MARTIN SILBERBERG AND RUTH SILBERBERG
Snodgras Laboratory, City Hospital, and Laboratory of the Jewish Hospital, St. Louis, Missouri[1]

THREE PLATES (NINE FIGURES)

The skeletal effects of the male sex hormone have been studied by administration of testosterone or by removal of the testicles. The results of these investigations, which are not in agreement, have been reviewed by Silberberg and Silberberg ('43), and by Simpson, Marx, Becks and Evans ('44). In immature female mice and rats, testosterone propionate accelerated skeletal development and ageing, whereas in immature guinea pigs removal of the sex glands delayed these processes. In growing rats and chickens, stunting was reported as well as a lack of effect on body growth. In rats and guinea pigs removal of the testicles was followed by a decrease in the growth rate. In castrate and in hypophysectomized young rats, small doses of the hormone caused a resumption of growth, and in stunted hypogenital boys growth could be stimulated by injections of testosterone. On the other hand, large doses of the hormone inhibited growth also in castrate rats. The present investigation was undertaken in order to obtain further insight into the role played by the male sex hormone in skeletal development and ageing.

MATERIAL AND METHODS

One hundred and ten mice of the inbred strain RFI, 4 weeks old at the beginning of the experiment and kept on a standard

---

[1] Aided by the Louis M. Monheimer Memorial Fund.

diet of Purina Dog Chow and water, were used. Seventy-seven animals were males and thirty-three females. The animals were divided into the following groups: I. Normal male animals (7 mice); II. Male animals with intact testicles injected subcutaneously with varying doses of testosterone propionate in sesame oil (33 mice); III. Males castrated at the age of 2 to 3 weeks (7 mice); IV. Castrated males treated with varying doses of testosterone (30 mice); V. Normal female animals (7 mice); VI. Female animals with intact ovaries injected with varying doses of testosterone (12 mice); VII. Females ovariectomized at the age of 2 to 3 weeks (7 mice); VIII. Ovariectomized females treated with testosterone (7 mice).

The hormone was administered in the following doses: 0.2 mg once weekly, 0.4 mg twice weekly, and 1 mg three times weekly. The periods of observation ranged from 4 days to 6 months. Details concerning the number of animals in each group, the dose administered,[2] and the duration of the experiment are given in the following table. At autopsy the lower end of the femur and the upper end of the tibia were taken out as a whole and sections were prepared for histological study.

HISTOLOGICAL EXAMINATION

A. In male mice

I. Growth zones. In the non-injected animal 8 weeks old (fig. 1), regular cartilage columns were separated from one another by thin sheets of ground substance. In an individual cartilage cell row there were eight to nine columnar and three hypertrophic cartilage cells. The latter underwent gradual calcification and were eventually eroded by bone marrow.

After injections of testosterone, the structural age of the epiphyseal plates was advanced beyond that seen in untreated animals of the same age, proliferation and hypertrophy were decreased, but hyalinization, calcification and ossification of the cartilage were increased. Larger doses given over short periods of time were more effective than prolonged treatment with smaller doses. Administration of 0.2 mg of testosterone once a week showed only a slight effect after 1 month of treatment. Injections of 0.4 mg twice weekly had a definite influence, which was evident as early as after 2 weeks, but

---

[2] We are indebted to Dr. Erwin Schwenk of the Schering Corporation for the generous supply of oreton.

EXPERIMENT	NO. ANIMALS	4 DAYS	1 WEEK	2 WEEKS	1 MONTH	2 MONTHS	3 MONTHS	6 MONTHS
Normal males	7	1	1	1	1	1	1	1
Normal males injected with 0.2 mg once weekly	14	2	2	2	2	2	2	2
Normal males injected with 0.4 mg twice weekly	14	2	2	2	2	2	2	2
Normal males injected with 1 mg three times weekly	5	1	1	1	1	1		
Castrated males	7	1	1	1	1	1	1	1
Castrated males injected with 0.2 mg once weekly	14	2	2	2	2	2	2	2
Castrated males injected with 0.4 mg twice weekly	14	2	2	2	2	2	2	2
Castrated males injected with 1 mg three times weekly	2				2			
Normal females	7	1	1	1	1	1	1	1
Normal females injected with 0.2 mg once weekly	7	1	1	1	1	1	1	1
Normal females injected with 1 mg three times weekly	5	1	1	1	1	1		
Ovariectomized	7	1	1	1	1	1	1	1
Ovariectomized injected with 0.2 mg once weekly	7	1	1	1	1	1	1	1

more so after 1 month. In animals receiving 1 mg three times, similar findings were already noticeable after 4 days. After 1 month of injections (fig. 2), the growth zones were only half as wide as in the untreated animal (fig. 1), the shortened cartilage cell rows contained only five or six columnar and two cells of hypertrophic type. The interstitial matrix was increased in amount and intensively hyalinized. Proliferation of the cartilage cells was almost at a standstill; the largest hypertrophic cells were much smaller and their breakdown was far less accentuated than ordinarily. A transverse bony lamella sealed the epiphyseal cartilage off from the metaphysis. A comparable condition was not seen in untreated mice before the age of 6 months.

In the castrate animal 8 weeks old (fig. 3) the epiphyseal plate was somewhat wider than in the normal control. The epiphyseal cartilage showed regular columns, each composed of ten to twelve columnar and three hypertrophic cells. The cartilage cells were smaller and the matrix was scantier than usual. The conversion of columnar into hypertrophic cartilage cells was more graded and the erosion of the calcified cartilage more irregular than ordinarily.

In the castrates receiving the hormone, the appearance of the epiphyseal disks depended again upon the dose of hormone given. After treatment with 0.2 mg of testosterone once weekly, for from 4 days to 2 weeks, little effect was noted. After 1 month of such injections (fig. 4), the cellular pattern was regular. A single cartilage cell row was composed of seven or eight columnar and two or three hypertrophic cells and it was separated from the adjoining rows by moderate amounts of matrix. The transition of columnar into hypertrophic cartilage cells was again more sudden, as seen in normal mice; the latter cells underwent calcification and were broken down by well filled capillaries. After administration of 0.4 mg of testosterone twice weekly, the effects were already visible after 2 weeks, when the proliferation of the columnar cartilage reached its peak. After hormonal treatment with this dose for 1 month, the proliferation of the cartilage was less accentuated than after injections of the smaller dose. In a single cartilage cell row seven columnar and two or three hypertrophic cells were counted. After administration of 1 mg three times weekly for 1 month (fig. 5), the growth zones were greatly narrowed, as compared with those of the untreated castrates; particularly the hyalinization of the cartilaginous matrix had increased. The cartilage cell rows were short and composed of five or six columnar and two cells of the hypertrophic type. Proliferation of the cartilage was decreased and calcification was adequate. The findings were thus comparable to those seen in animals with intact testicles but receiving a smaller dose of the hormone.

The following photomicrographs (figs. 6–9) illustrate the conditions in mice 4 months of age. In the untreated animal (fig. 6) the growth zone was narrow but still showed a regular arrangement of the cartilaginous columns. The cartilaginous matrix was dense. A single cartilage cell row contained five or six inactive columnar and two small cells of hypertrophic type; however the difference between these two kinds of cells began to disappear. Here and there, individual cartilage cells showed signs of disintegration and a bony lamella underneath the cartilage began to seal off the cartilage from the marrow.

In 4 months old mice having received 0.2 mg of the hormone once a week for 3 months, little deviation from the normal was noted. After treatment with 0.4 mg of testosterone twice weekly for 3 months (fig. 7), the epiphyseal disk was irregular, hyalinized and more calcified than usual; the cartilaginous matrix was abundant, and numerous thick plugs of amorphous material were present in the growth zone. In areas in which calcification did not mask the cellular details, a single cell row consisted of five or six cells; differences between columnar and hypertrophic cells were hardly noticeable. The epiphyseal cartilage was delimited from the bone marrow by a continuous transverse osseous plate.

In the castrate (fig. 8), the epiphyseal disk was somewhat wider and the cartilaginous matrix was scantier than in the normal control. A cartilage cell row contained seven or ten columnar cells; the number of the hypertrophic cells was so reduced that no accurate counts could be made. On the whole, the epiphyseal plate showed a more youthful appearance than that of the normal control.

Again in the castrate animal, 0.2 mg of testosterone, given one weekly for 3 months, was unable to produce noticeable changes. However, 0.4 mg of the hormone given to castrates three times weekly for 3 months (fig. 9) caused marked hyalinization, calcification, and some disintegration of the cartilage. Due to these regressive changes, exact cell counts could not be obtained. The structural age of the growth zones was thus somewhat advanced beyond that seen in normal mice of the same age.

In animals 7 months old observed for a period of 6 months the histological findings were as follows: In untreated mice the growth zones were composed of inactive hyalinized and calcified cartilage showing plugs of disintegrated cells. The cartilage was sealed off from the bone marrow by a transverse bony plate.

In the animal treated with 0.2 mg of testosterone once a week, conditions did not differ from those seen in the untreated control. Under the influence of 0.4 mg of the hormone, administered twice a week, the epiphyseal cartilage was heavily calcified; plugs of destroyed cartilage were ossified, and they were more numerous and thicker than in the normal control. The bony lamella delimiting the cartilage from the marrow was thinner than usual and capillaries began to dissolve the osseous plugs, thus indicating impending perforations. This did not occur in untreated controls before the end of the first year of life.

In the castrate, the hyalinization and calcification of the cartilage were less accentuated than in the normal animal, and the plugs traversing the epiphyseal plate were fewer in number and smaller in size than in the normal control.

Orchidectomized animals treated with 0.2 mg of testosterone once weekly showed no hormonal effects. Administration of 0.4 mg of the hormone twice weekly over this prolonged period of time produced a structural age of the growth zone comparable to that seen in untreated mice of corresponding age.

II. Diaphysis. In normal mice 4 to 8 weeks old (fig. 1), osteoblasts were scarce in the proximal, but more numerous in the distal metaphysis. The trabeculae were thin and contained moderate amounts of calcium. The shafts were covered by loose periosteum containing some osteoclasts.

Administration of 0.2 mg of testosterone once a week for from 4 days to 2 weeks was ineffective. After 2 and 4 weeks of injections of 0.4 mg twice a week, some effects were noticeable, but under the influence of 1 mg of the hormone given three times weekly, the changes were already conspicuous after 1 week; they were even more pronounced after 1 month. There was a poorly vascularized connective tissue in the metaphysis, with numerous osteoblasts packed closely together. The spicules were thickened and short and connected with one another by transverse bony bridges (fig. 2). Farther distally, the bone marrow showed its usual structure; however, deep in the diaphysis there were numerous remnants of spicules that had not been completely absorbed. The shaft was thicker than in the untreated controls and contained small osteocytes and much calcium. The periosteum was dense, the channels perforating the shaft were narrower than in the untreated controls and filled with connective tissue and hemopoietic marrow.

During the first month following castration the metaphyseal capillaries were collapsed and a loose connective tissue was found underneath the epiphyseal cartilage. There was no bony lamella underneath the cartilage (fig. 3). Here and there, strands of calcified uneroded cartilage cells were incorporated in the spicules. The trabeculae were more delicate and slender than in the normal animal, the shaft was less calcified than usual and it contained numerous vessels and large osteocytes.

In castrates treated with testosterone, the structure of the metaphysis changed with the dose of the hormone given. Administration of 0.2 mg once a week exerted a slight effect after 2 weeks and a more marked one after 4 weeks (fig. 4). The subepiphyseal tissue was then better vascularized than in the untreated castrate and more osteoblasts were laid down along and in the trabeculae. After injections of 0.4 mg of the hormone twice a week, increased vascularization of the metaphysis was noted already after 2 weeks, and after 1 month the structure of the metaphysis resembled more closely that seen in untreated mice of the same age. The vascularization was good and fairly long calcified spicules were formed; the number of osteoblasts and the resorption of the primary bone were within normal limits. The periosteum and the shaft also appeared normal. After treatment with 1 mg of testosterone three times weekly, changes were definite as early as after 4 days. After 4 weeks (fig. 5) a thin, discontinuous bony lamella was present underneath the hypertrophic cartilage. The trabeculae contained abundant calcium and showed some interlacing. The connective tissue along the spicules was dense, many osteoblasts were visible, but resorption of bone was more marked than under the influence of the smaller doses. The spicules were then short and thick. The shaft contained much calcium and numerous small osteocytes. The periosteum was denser than in the untreated castrate or in the castrate that had received the smaller dose of the hormone.

In 4 months old controls (fig. 6), a bony lamella sealing off the epiphyseal cartilage from the metaphysis was being formed and the spicules were thick and long. The shaft consisted of small osteocytes and much ground substance. The periosteum was denser and the vascularization was less marked than earlier in life.

After 3 months of injections of 0.2 mg of the hormone the change was not as pronounced as after administration of 0.4 mg twice a week. Under the latter condition (fig. 7), there was a thick transverse bony plate underneath the epiphyseal cartilage. This plate was eroded by bone marrow. The bone was markedly calcified and the trabeculae formed an interlacing network; resorption of the latter was progressing. Likewise, the periosteum showed intensified formation and destruction of bone.

Three months subsequent to castration (fig. 8), a thin bony plate delimited the epiphyseal cartilage from the bone marrow. The trabeculae were thin and slender.

In the castrate treated with 0.2 mg of the hormone for 3 months, the changes were negligible. In animals receiving 0.4 mg twice a week (fig. 9), a thick, but still discontinuous, transverse bony lamella was found under the epiphyseal cartilage; the primary bone was resorbed. The periosteum was denser than in the untreated castrate.

In normal mice 6 to 7 months old, some short trabeculae were still present in the metaphysis. In the treated animal of corresponding age, most of the primary spicules were dissolved. In castrates of this age the trabecular bone was more abundant than in the animal with intact testicles. In the castrate receiving the hormone, conditions were almost comparable to those seen in the normal control animal.

B. In female mice

I. Growth zones. During the first 4 months of life the age changes in the epiphyseal cartilage were somewhat advanced as compared with those seen in the males of the same age. In the untreated female, 2 months old, the regular cartilage cell rows were separated from one another by moderate amounts of chondromucoid matrix. In a single row, eight columnar and three hypertrophic cells were counted.

Previously ('41), we reported on the effect of testosterone in growing female mice. The present experiments substantiate our former observations. In brief, the administration of testosterone intensified the age changes in the epiphyseal cartilage. Animals 2 months of age and treated with 0.4 mg of testosterone twice weekly showed a growth zone whose structural age was about 2 months advanced beyond its chronological age.

In spayed mice, 2 months old, the epiphyseal disk was wider, the cartilaginous ground substance scantier and less calcified than in the normal control. The individual cartilage cell row was composed of ten columnar and four hypertrophic cells, the latter being very large. There was no sealing off of the cartilage from the metaphysis. These findings resemble those seen in normal mice 4 to 6 weeks of age.

In ovariectomized animals, the administration of 0.2 mg of testosterone once weekly was somewhat more effective than in the castrate males. After 1 month of treatment, the growth zones contained more calcium than in the untreated spayed mouse. The cartilaginous matrix was abundant, and in the single cartilage cell row seven columnar cells and only one hypertrophic cell were counted. Hypertrophy was thus far less accentuated than in the spayed non-injected mouse. Plugs of degenerated cartilage were not found nor was a transverse bony lamella laid down. The histological age of the growth zone of a 2-month-old female was thus comparable to that of an untreated control about 3 months old. The male castrate showed hardly any response to the same treatment.

After 3 months of observation similar differences were noticeable in the various groups. In the untreated control, 4 months of age, the epiphyseal disk was still regular and only moderately calcified, a single cartilage cell row containing five or six inactive columnar and two relatively small cells of the hypertrophic type. The cartilaginous matrix was scanty and here and there a cartilage cell underwent destruction. Underneath the cartilage a transverse bony lamella began to form.

Three months after ovariectomy the epiphyseal disk of a mouse, then 4 months old, resembled that of an untreated animal 2 to 3 months of age. The cartilage cell rows consisted of nine columnar and three hypertrophic cells. There was only moderate calcification, disintegration of cartilage was lacking, and no osseous plate was seen underneath the hypertrophic cartilage.

Administration of 0.2 mg of the hormone once a week for 3 months altered the condition noted after ovariectomy in the following way: A cartilage cell row was now composed of five columnar and two hypertrophic cells. The matrix was more abundant and calcification more intensive; moreover, a few plugs of disintegrated cartilage were found in the growth zones. These age changes were, however, less advanced than in the non-spayed mouse receiving corresponding doses of testosterone. Thus again, the epiphyseal plate in the spayed animal treated with hormone resembled that of the normal animal of the same age.

II. Diaphysis. In 8-week-old mice treated with 0.2 mg of testosterone for 4 weeks, the metaphysis contained more trabecular bone than in the untreated control, and the spicules showed a slight tendency to interlacing. At the shaft the periosteal connective tissue was more abundant and denser than usual.

In the ovariectomized animal of corresponding age, the spicules were longer and thinner than in the untreated control, whereas in the spayed animal receiving 0.2 mg of the hormone for 4 weeks, more and thicker bone was found than without hormonal treatment.

In the animal observed for 3 months after ovariectomy, long and slender spicules were present in the metaphysis. 0.2 mg of testosterone given weekly to the spayed animal for a period of 3 months, produced a more normal appearance of the remaining osseous spicules.

COMMENT

In immature male mice testosterone propionate, in direct proportion to the dose administered, accelerated ageing of the epiphyseal cartilage by decreasing proliferation and by increasing regressive changes; it diminished somewhat the resorptive activity of the bone marrow. These effects in males were similar, although less marked than in immature female mice. The ageing effect of testosterone on the epiphyseal cartilage of hypophysectomized growing rats, treated simultaneously with anterior hypophyseal hormone, has been shown recently by Reiss, Fernandez and Golla ('46).

Castration performed in immature male mice, delayed the development of the epiphyseal cartilage by decreasing the regression and by inhibiting the hypertrophy of the cells, while the proliferation of the cartilage went on to a limited degree. Thus, the individual cells and the growth zones as a whole retained a youthful appearance for an abnormally long period of time. In the metaphysis a temporary mild fibrosis occurred, associated with the production of delicate and short trabeculae. In female mice, spaying retarded the ageing of the epiphyseal cartilage by diminishing its regression. However, the development of the cartilage, as indicated by its hypertrophy, was not inhibited as it was in the castrate males, and while the primary spicules also were slender, there was no fibrosis in the metaphysis. The findings in castrate male and female mice thus resembled those seen in castrate male and female guinea pigs (Silberberg and Silberberg, '43). As in the latter, the changes following castration were only of a temporary nature.

Administration of small or moderate amounts of testosterone to castrate male or female mice was followed by resumption of the regular development and ageing of the epiphyseal cartilage. In males the normal rate of proliferation of the columnar cartilage cells was restored and a more rapid conversion of the latter into hypertrophic cells took place. In both sexes, regression of the cartilage was increased beyond conditions seen in untreated castrates and the trabeculae became coarser and longer. If the amount of hormone given exceeded the substitution level, the changes resembled those seen in the noncastrate mice receiving the smaller dose of the hormone. These findings in mice are in agreement with the effects of large and small doses of testosterone on body length of castrate rats (Rubinstein and Solomon, '41).

There is, then, the apparently paradoxical phenomenon that the administration of testosterone as well as the removal of the testicles resulted in an inhibition of growth. However, two different mechanisms accounted for this occurrence: The inhibition of growth seen under the influence of testosterone is a direct consequence of the suppression of the proliferation of the cartilage cells. It has been suggested that this effect may be due to an inhibition of the anterior hypophysis by the testosterone. However, this hormone exerts skeletal effects also in hypophysectomized animals (Simpson, Marx, Becks and Evans, '44; Reiss, Fernandez and Golla, '46). It is therefore probable that testosterone likewise acts directly on the cartilage in animals with intact pituitaries. Following removal of the testicles, on the other hand, the hypertrophy of the cartilage is incomplete and its breakdown is therefore slow. This disturbance of development is followed by changes in the rate of growth. If proliferation of the cartilage cells went on undisturbed, while resorption was decreased, the epiphyseal plates would necessarily grow wider and wider. This does not occur however. It must therefore be assumed that the proliferation of the cartilage is likewise slowed down. Previously, we attributed the increased number of columnar cartilage cells seen in castrate male animals to an active proliferation, possibly due to a stimulation of the anterior hypophysis. In the light of the recent experiments, however, it seems more likely that the increased number of columnar cartilage cells represents merely a crowding of the cells in the growth zones, due to both insufficient development and breakdown.

Administration of testosterone to castrates reversed the retardation of skeletal development caused by orchidectomy and thus, as in noncastrate animals, accelerated skeletal maturation and ageing. Following the restitution of normal development of the cartilage the usual rate of proliferation is restored. The growth-promoting effect of testosterone in the castrate or hypogenital individual is then only incidental to the resumption of skeletal development and not a primary effect on the cartilage. This seems to be corroborated by the fact that skeletal growth cannot be stimulated beyond the normal, even if high doses of testosterone are given to the castrate animal.

There are sex differences in the response of the epiphyseal cartilage of mice to removal of the sex glands. Unlike in the male castrate, in the spayed female the hypertrophy of the cartilage was not inhibited; and there was active breakdown of the large hypertrophic cartilage cells, and no fibrosis in the metaphysis. Thus, the cartilage of the ovariectomized animal developed in a more normal fashion, that is, more rapidly and more completely than that of the orchidectomized male. This may be considered as an expression of the genetically determined faster rate of skeletal ageing in the female, which manifests itself also in human fetuses and children (Hill, '39; Francis, '40; Greulich, '42). Moreover, the bones of the spayed female guinea pigs and rats grow better than those of castrate males (Moore, '22; Tang, '41). It remains undecided whether these genetic differences concern only the cartilage; genetically determined differences in the action on the skeleton of endocrines other than the sex glands may also play a role.

Testosterone counteracted the skeletal changes caused by castration in female as well as in male mice. Moreover, in guinea pigs the effects of castration on cartilage and bone could be inhibited by administration of anterior hypophyseal hormone (Silberberg and Silberberg, '43). Thus the effects of castration and probably of certain non-ovarian hormones on the growth of the cartilage are largely nonspecific in the sense that any inhibitor can be counteracted by any activator provided the two act on the same substratum. This conclusion seems to be borne out also by the fact that testosterone can at least partly reverse the effects of hypophysectomy on the skeletal tissues of growing rats (Simpson, Marx, Becks and Evans, '44).

SUMMARY

In immature male mice, testosterone propionate accelerated the ageing of the epiphyseal cartilage by inhibiting proliferation and by increasing regressive changes. These effects were less marked in males than in immature females. Removal of the testicles caused a delay in the development of the cartilage, followed by a reduction in the rate of proliferation. This decrease of proliferation represents the direct cause of the temporary reduction in body length seen in castrate animals.

Administration of small doses of testosterone propionate to castrate mice restored both development and growth of the epiphyseal cartilage. The resumption of growth is incidental to the restitution of the development of the cartilage and is not due to a direct growth-stimulating effect of the hormone.

LITERATURE CITED

FRANCIS, C. C. 1940 The appearance of centers of ossification from 6 to 15 years. Am. J. Phys. Anthrop., vol. 27, p. 127.

GREULICH, W. W. 1942 Genic and endocrine determination of skeletal development. Endocrinology, vol. 30, p. S. 1023.

HILL, A. H. 1939 Fetal age assessment by centers of ossification. Time table for the appearance of centers of ossification in consecutive months of fetal life on the basis of 50% incidence. Am. J. Phys. Anthrop., vol. 24, p. 251.

MOORE, C. R. 1922 On the physiological properties of the gonads as controllers of somatic and psychial characteristics. V. The effects of gonadectomy in guinea pigs on growth, bone length and weight of organs of internal secretion. Biol. Bull., vol. 43, p. 285.

REISS, M., J. E. FERNANDEZ AND Y. M. L. GOLLA 1946 The peripheral inhibitory influence of large doses of testosterone on epiphyseal cartilaginous growth. Endocrinology, vol. 38, p. 65.

RUBINSTEIN, H. S., AND M. L. SOLOMON 1941 The growth depressing effect of large doses of testosterone propionate in the castrate albino rat. Endocrinology, vol. 28, p. 112.

SILBERBERG, M., AND R. SILBERBERG 1941 Response of cartilage and bone of growing mice to testosterone propionate. Arch., Pathol., vol. 32, p. 85.

———— 1943 Influence of the endocrine glands on growth and aging of the skeleton. Arch. Pathol., vol. 36, p. 512.

SIMPSON, M. E., W. MARX, H. BECKS, AND H. M. EVANS 1944 Effect of testosterone propionate on the body weight and skeletal system of hypophysectomized rats. Synergism with pituitary growth hormone. Endocrinology, vol. 35, p. 309.

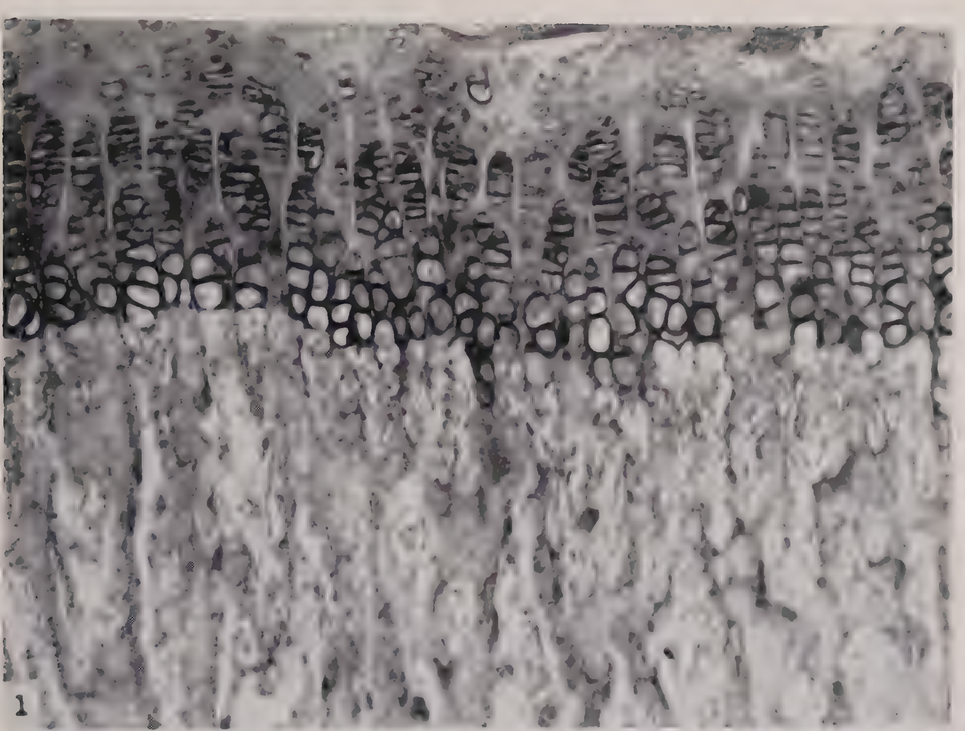
TANG, Y. Z. 1941 Sex differences in growth in gonadectomized rats. Anat. Rec., vol. 80, p. 13.

PLATE 1

EXPLANATION OF FIGURES

1 Section through the growth zone of the upper tibia of a normal male mouse of strain RFI, 8 weeks old. There is regular arrangement of the cartilage cell rows, breakdown of the hypertrophic cartilage cells, and formation of bony spicules are in progress. 175 ×.

2 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 8 weeks old and injected with 1 mg of testosterone propionate three times weekly for 4 weeks. The growth zone is greatly narrowed, both columnar and hypertrophic cartilage cells are decreased in number and smaller than usual (fig. 1). The interstitial matrix is markedly increased in amount. The metaphyseal spicules are short and begin to form a transverse lamella. 175 ×.


1

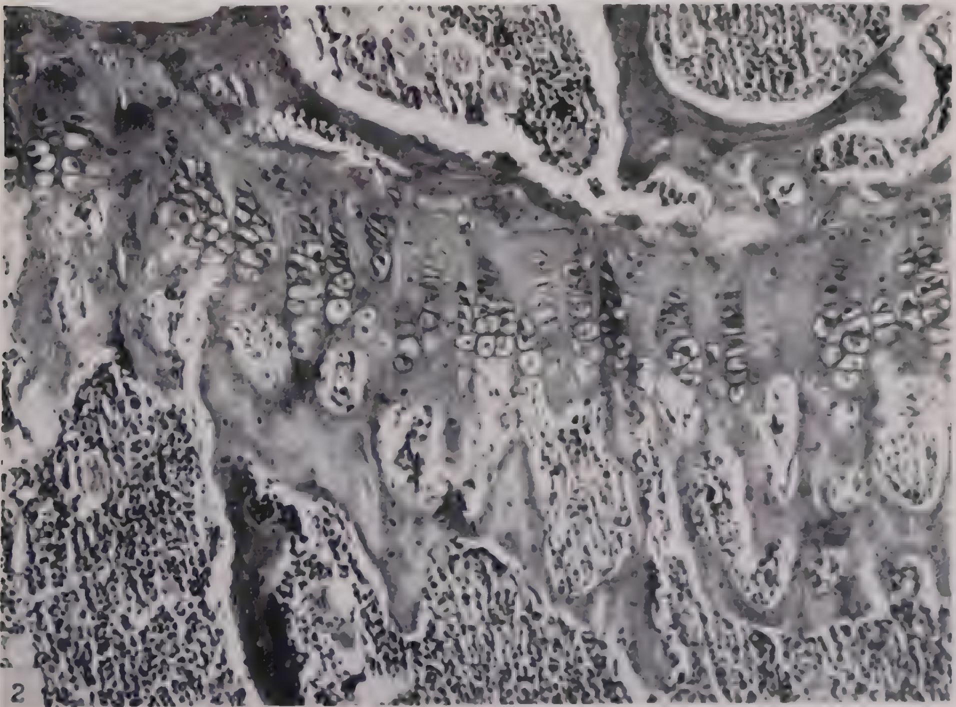

2

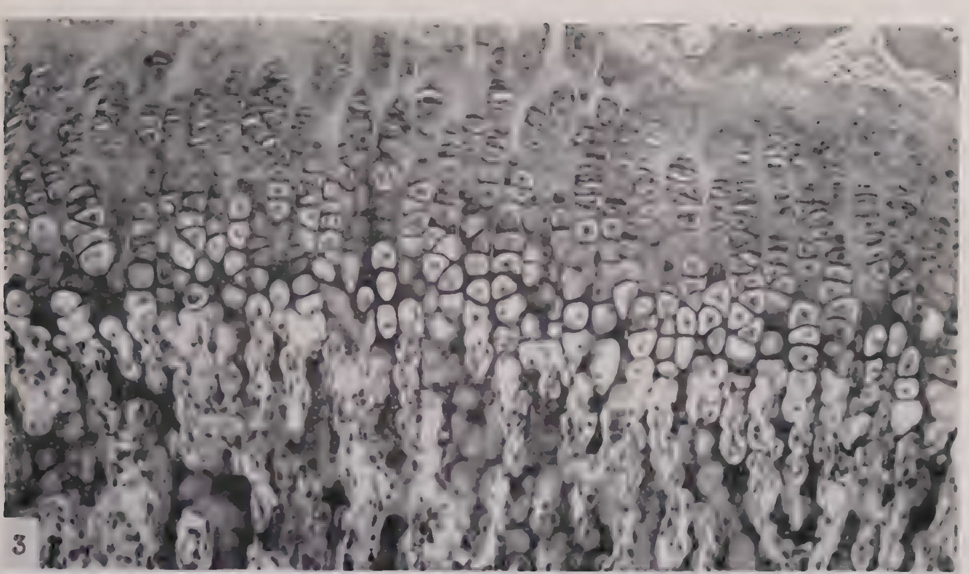
PLATE 2

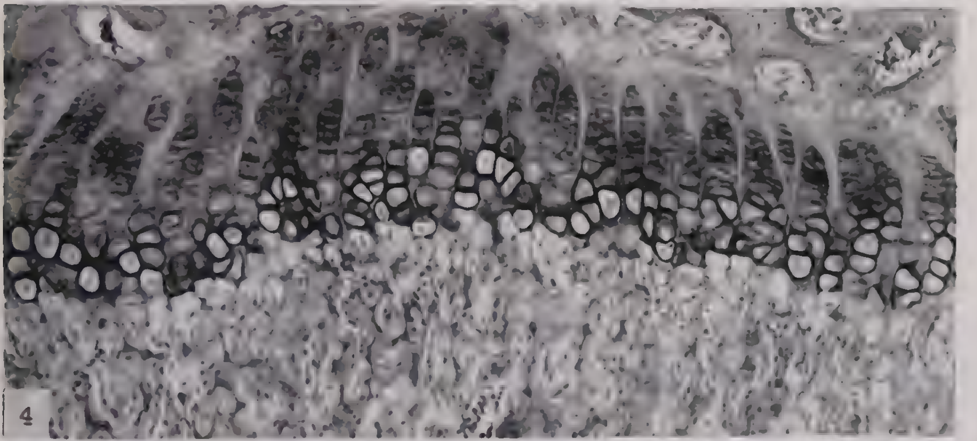
EXPLANATION OF FIGURES

3 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 8 weeks old and castrated at the age of 4 weeks. There is a crowding of cartilage cells in the epiphyseal plate; the hypertrophic cartilage cells do not reach the same size as the corresponding cells in the control mouse (fig. 1); little interstitial substance is present; the osseous spicules are thin and delicate. 175 ×.

4 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 8 weeks old, castrated at the age of 4 weeks and injected with 0.2 mg of testosterone propionate once weekly for 4 weeks. The growth zone is narrower and there are fewer cells and more matrix than after castration alone (fig. 3). The structural age of the epiphyseal disk resembles more that of the normal control (fig. 1). 175 ×.

5 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 8 weeks old, castrated at the age of 4 weeks and injected with 1 mg of testosterone propionate three times weekly for 4 weeks. The growth zone is somewhat narrower and more hyalinized and there are fewer cartilage cells than in the normal animal (fig. 1). The trabeculae are more mature than in the latter. 175 ×.


3


4

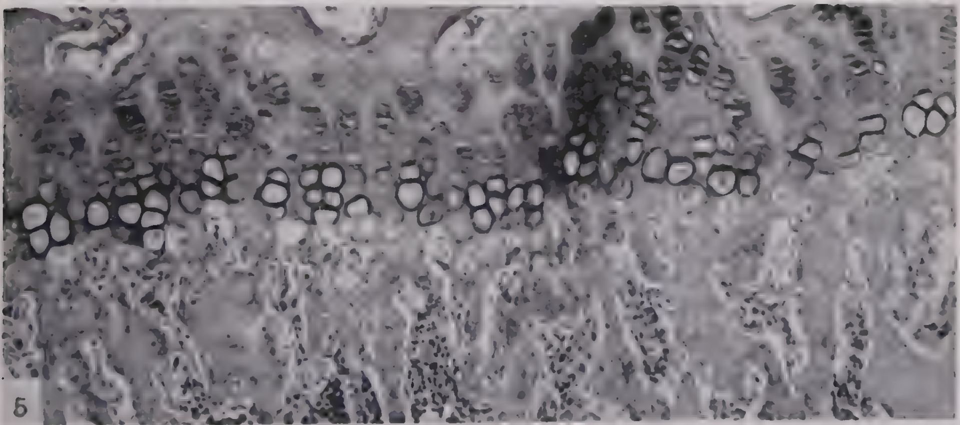

5

PLATE 3

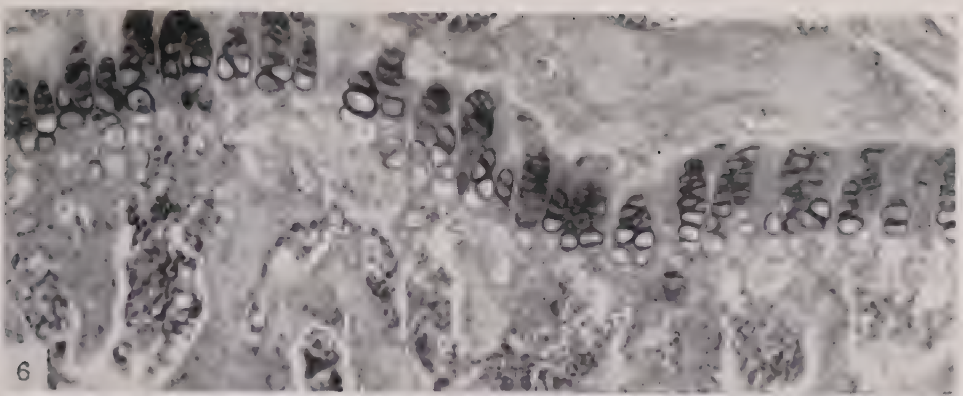
EXPLANATION OF FIGURES

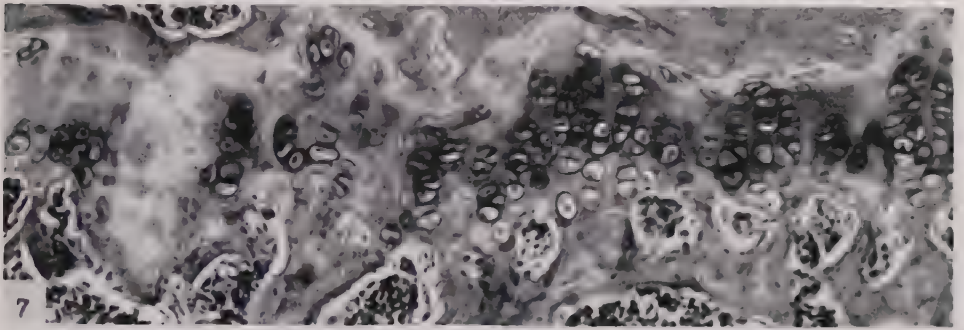
6 Section through the growth zone of the upper tibia of a normal male mouse of strain RFI, 4 months of age. The growth zone is narrow, regularly configurated but shows a good deal of hyalinization; there is abundant bone in the metaphysis. 175 ×.

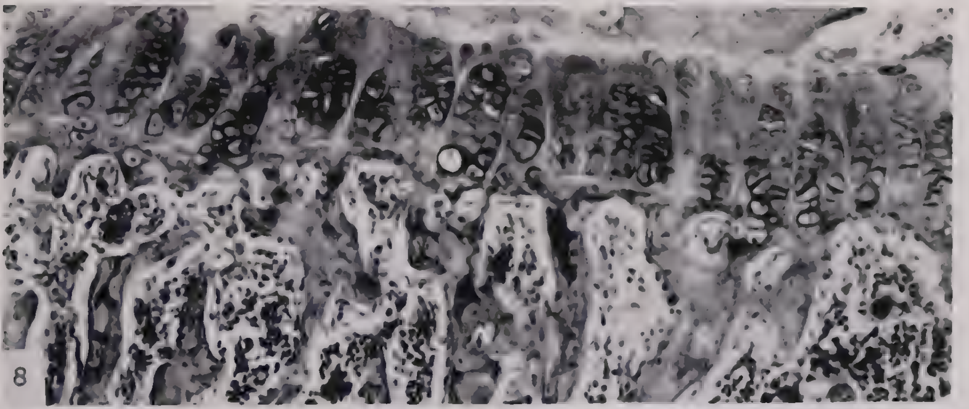
7 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 4 months of age and injected with 0.4 mg of testosterone propionate twice weekly for 3 months. There is widespread degeneration of the inactive cartilage; a thick bony plate traverses the metaphysis. 175 ×.

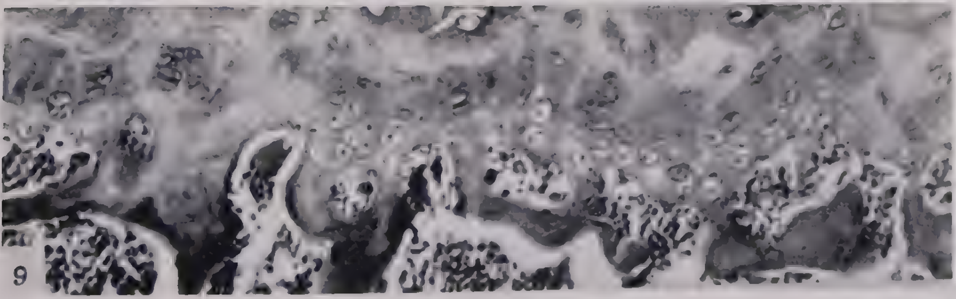
8 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 4 months of age and castrated at the age of 4 weeks. The cartilage cells in the epiphysis are more numerous and less hyalinized than in the normal control (fig. 6). Hypertrophic cartilage cells are scarce and the trabeculae are delicate. 175 ×.

9 Section through the growth zone of the upper tibia of a male mouse of strain RFI, 4 months of age, castrated at the age of 4 weeks and injected with 0.4 mg of testosterone propionate twice weekly for 3 months. Hyalinization of the cartilage is marked. The metaphyseal bony lamella is thinner than in the non-castrate animal treated with corresponding amounts of testosterone propionate (fig. 7). 175 ×.


6


7


8


9

METHOD OF DUPLICATING ANATOMICAL SPECIMENS

CAPTAIN LOUIS EMORY
Dental Corps, Army of the United States

EIGHT FIGURES

In the course of training technicians for the Medical Department, it has been necessary to use a great number of anatomic models as training aids. The study of anatomy is facilitated by using actual anatomic specimens; however, these are limited in number and models must be substituted whenever possible. These models must resemble the specimen in all details, otherwise they will confuse rather than aid the student.

Duplicating requires special materials and many were tried before one was found which met all of the requirements. With this material, it is possible to reproduce minute details so that the reproductions may be distinguished from the original specimen only with difficulty. The molds are permanent and can be used many times with no loss of detail. The material has sufficient elasticity to permit withdrawal from large undercut areas. It can be made thick enough to support itself without the use of a matrix; if needed, a plaster of Paris support may be used.

The use of this material and technique has been limited (although not necessarily) to reproductions of the skull, mandible and teeth and the resulting models used at the Medical, Surgical, X-ray and Dental Sections of the Med. Dept. Enl. Tech. School at Billings General Hospital. It is possible that this technique may be of value not only for the reproduction of musuem specimens, but also for repairing and establishing permanent records of these specimens.

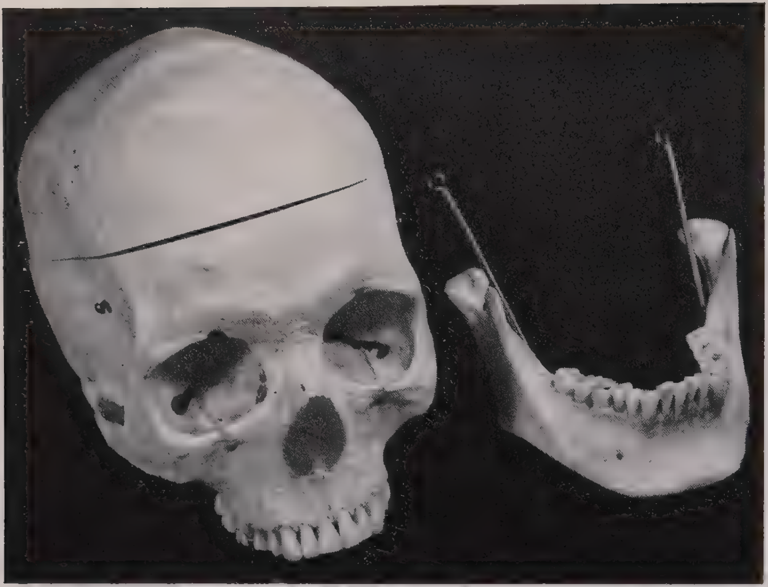


Figure 1

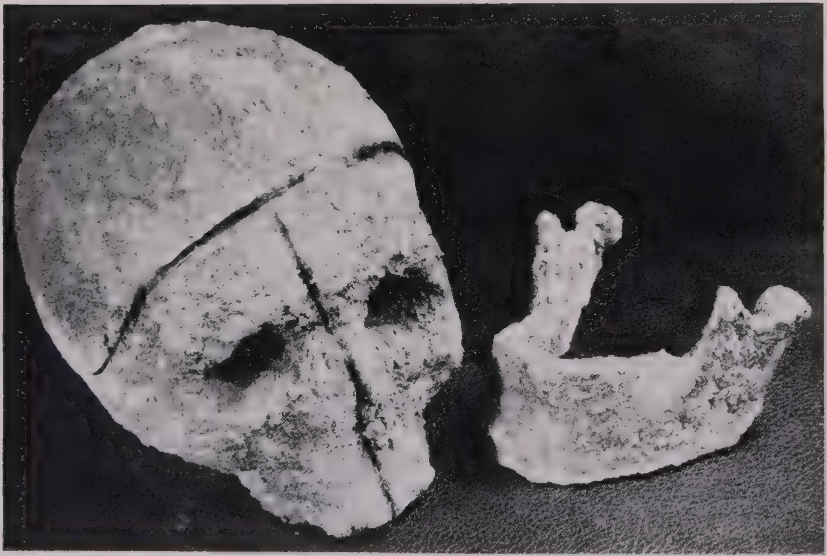


Figure 2

The mold consists of an ammonical liquid latex[1] solution and this is supported by a heavier coat of plastic material which cures to the liquid rubber portion to form the elastic mold proper. This is supported by other means if a more rigid mold is necessary. Plaster of Paris, strips of burlap or sheets of gauze incorporated into the plastic material may be used for additional reinforcement.

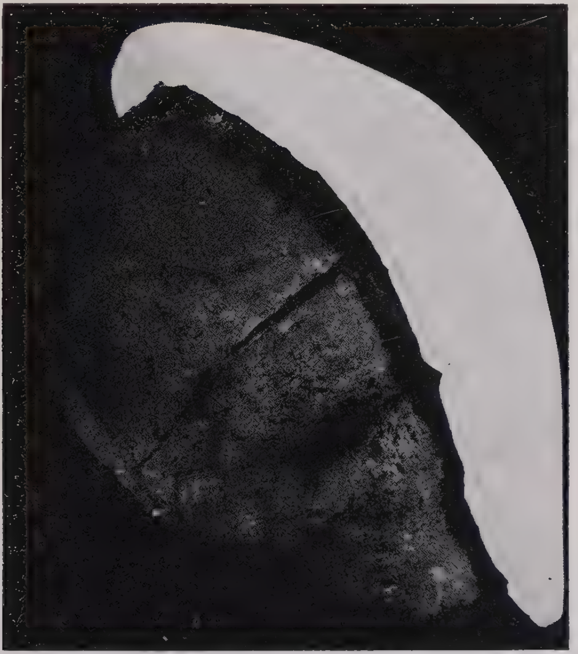


Figure 3

Figure 1 shows the original skull and mandible with the preparation that is required before starting the mold. No separating medium is required and areas such as the zygomatic arches and the nares are blocked with wax to facilitate separation of mold from the skull. The liquid rubber solution is painted on in thin coats and permitted to cure before subsequent coats are applied. This is then reinforced with a thick

---

[1] So-Lo-Go-Ma, Solo Works, Cincinnati, Ohio.

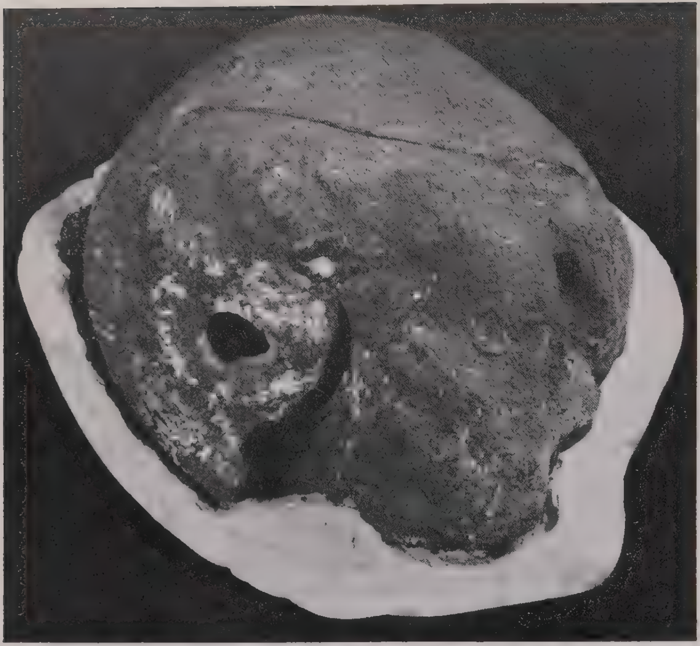


Figure 4

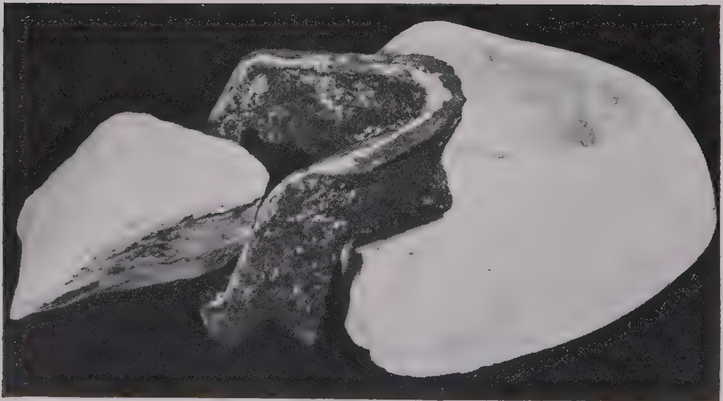


Figure 5

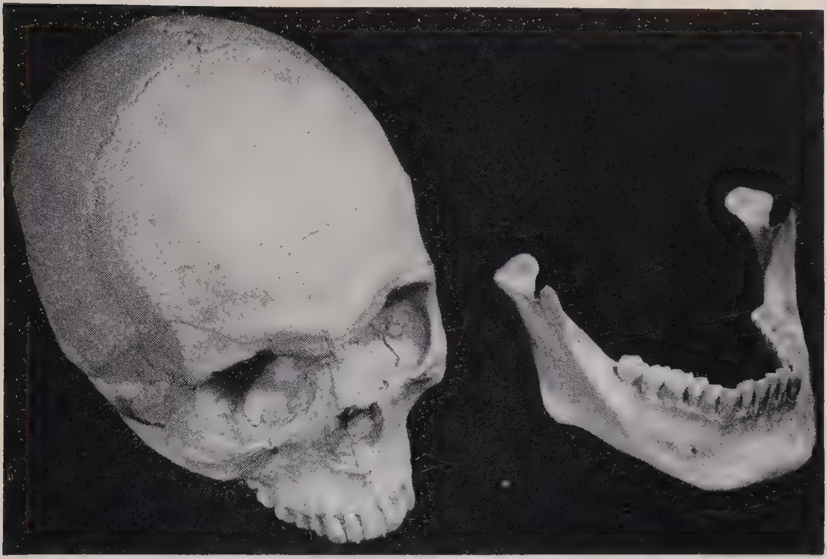


Figure 6

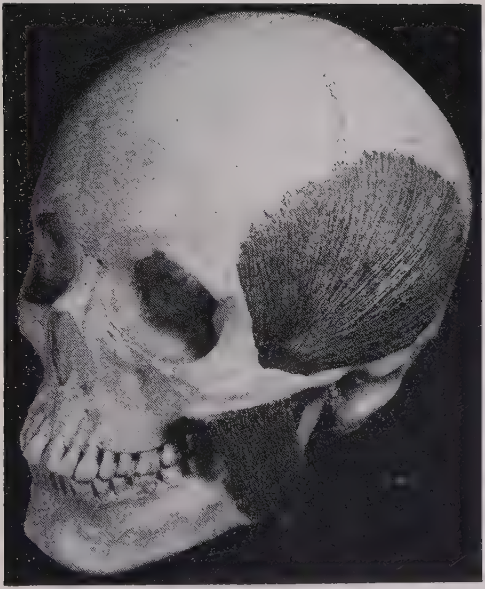


Figure 7

coat of the plastic supporting material which should be at least $\frac{1}{2}$ inch in thickness. For the mandible, the plastic supporting material need be only $\frac{1}{4}$ inch thick since it is well supported by a plaster of Paris matrix.

Figure 2 shows the material after it has cured and the lines on the skull model where the mold was cut for separating. Figures 3, 4, and 5 demonstrate the plaster matrix; however, the skull mold is thick enough to support itself.

The stone models produced with mold are shown in figure 6. The skull is made hollow. This is done by pouring dental stone of a creamy consistency into the mold. The excess is then poured off and this stone wash is allowed to approach the initial setting stage followed by thin washes of dental stone that are poured into the mold to give the model the desired thickness.

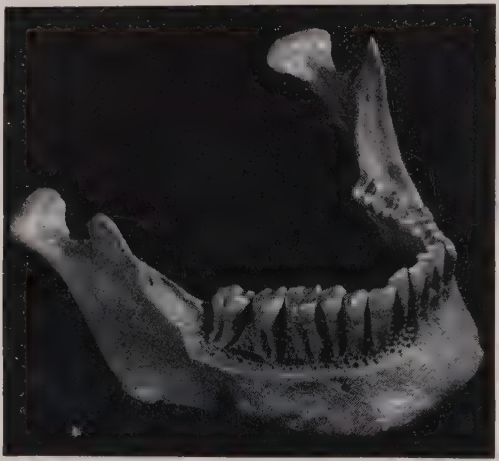


Figure 8

Figures 7 and 8 show some of the models that were made by this method. Figure 7 shows the masseter and temporalis muscles. A mandible with exposed roots of the teeth is shown in figure 8.

A COMPARISON OF GROWTH, DIFFERENTIATION, ACTIVITY AND ACTION CURRENTS OF HEART AND SKELETAL MUSCLE IN TISSUE CULTURE[1]

J. SZEPSENWOL[2]
Section of Neuro-Anatomy, Yale University School of Medicine,
New Haven, Connecticut

THREE FIGURES

INTRODUCTION

The heart and the skeletal muscles start their differentiation at a very early stage of development. Their activity is also precocious, but whether it precedes or follows the structural organization is not yet known. In the case of the myocardium, the organ, from the beginning, appears as an isolated entity. Its evolution, in relation to its functional activity, can be studied, therefore, without great difficulty in vivo as well as in vitro. Tissue culture technic is helpful since differentiation and activity of the heart in vitro follow, at least in the beginning, the same pattern as during normal development. The presumptive heart tissue starts to contract rhythmically and to differentiate into cross striated fibers even when isolated from the organism and cultivated in vitro. Moreover, electrical records taken from explants of the myocardium seem to be analogous to those of the normal adult heart.

---

[1] Aided by a grant from the Fluid Research Funds, Yale University School of Medicine.

[2] Knight Fellow, Section of Neuro-Anatomy, Yale University School of Medicine, January, 1944 to July, 1945.

The skeletal muscle, as it appears from studies made on the behavior of embryos of different species, becomes active relatively late. In the chick embryo the body musculature does not start to contract before the fourth day of incubation (Windle, '34; Kuo Zing Yang, '32). At that time the muscle fibers show cross striation, the nervous system is in active differentiation and nerves of varying size extend from the spinal cord to the myotomes. Whether there is any kind of muscular activity before that stage of development could not be established with certainty by purely morphological studies.

Many investigators attempted to cultivate in vitro somatic muscle of young embryos of different species; they generally failed to obtain any typical differentiation (Hogue and Renyi, '31). Some of them observed the transformation of myoblasts into cross-striated muscle fibers, but these fibers did not show any activity. Lewis ('20) did obtain the contraction of muscle fibers in tissue culture but, as the author himself recognizes, in his experiments the entire explant remained inactive.

Working with different culture media, the author ('41) found that under certain conditions explants of skeletal muscle of young chick and rat embryos may undergo normal differentiation and start to contract rhythmically. The activity of the muscle in such tissue cultures has been described previously ('41–'43). The present report is an attempt to compare the functional and morphological evolution of the skeletal muscle, as well as its action currents to those of the heart explants.

TECHNIC

The hanging drop technic was used. The culture medium was made of guinea pig plasma and embryo juice diluted with Ringer. In order to prevent the coagulation of the blood, while it was taken by cardiac puncture, Liquemin,[3] the Roche-Organon brand of heparin, was used. It is a very potent anticoagulant, 1 cm^3 of the original solution is enough to prevent the coagulation of a litre of blood, consequently it had to be used in low concentration. Generally, 1 cm^3 of a saline solution containing 1% of Liquemin was added to every 10 cm^3 of blood, which is the amount drawn at a time from a guinea pig of about 350 to 400 gm. In such a concentration, Liquemin does not seem to have any inhibiting effect upon the growth of the tissue; its anticoagulant property, which keeps the plasma in its liquid state for several days, is rapidly neutralized by the embryo juice.

---

[3] The author is indebted to the Roche-Organon Company for supplying the Liquemin.

The embryo juice, as usual, was made of chick embryos 8 to 9 days old. It was diluted with Ringer in the proportion of 1 to 50, or 1 to 100, since it was found that a medium poor in nutritive substances is favorable for the differentiation and activity of skeletal muscle.

The skeletal and heart muscle to be cultivated were isolated from chick embryos of different ages (1 day of incubation to hatching). The explants were kept in the same medium from 5 to 7 days and were then transferred to a new coagulum. Observations were made twice daily under the microscope, and electrical records were taken with the Grass amplifier.

In order to take the electrical records, the preparation had to be opened, turned upside down, and placed under the microscope on a warm stage (heated to 37–38°C.). Two silver electrodes, connected with the amplifier and moved with the aid of a micromanipulator, were put in contact with the coagulum, as close as possible to the explant. The distance between the electrodes and the explant, together with their orientation, seems to be of importance in the amplitude of the spikes. These are higher when the electrodes are close to the explant and are oriented in the direction of the muscle fibers.

RESULTS

In chick embryos the heart, as a single tube, appears after 1 day of incubation, and at the age of 33 to 35 hours it starts to pulsate. The moment of initiation of activity in the myocardium in tissue culture is the same as during normal development. The presumptive heart tissue of embryos 12 to 24 hours old was isolated and explanted in vitro. After 10 to 24 hours of growth it started to pulsate slowly, at the beginning, and at a rate of about one pulsation per second a little later.

An explant from an embryo 12 hours old often differentiates into two hearts, pulsating each with its own rhythm. The rate of pulsation of each of these hearts and the moment of initiation of activity are similar to those of a single normal heart. An explant from an embryo 24 hours old generally differentiates into a single heart which is small in size and abnormal in form. Even after 1 or 2 weeks of growth in vitro it does not attain the size of the heart of an embryo 2 days old, and the parts corresponding to the atrium and to the ventricle are not recognizable morphologically. The differentiation, however, proceeds normally, at least at the beginning. Cross-striated fibers appear in the culture 1 to 2 days after the explantation. They rapidly increase in number during the first week of culture. Later, degenerative changes make their appearance if the culture medium is not changed.

Of particular interest for the subject of this paper is the fact that, in spite of its abnormal form, the explant contracts from the beginning as a unit. The contraction starts generally at one point and propagates rapidly toward the rest of the explant. It is true that after 7 or 8 days of growth the different parts of the explant may start to contract separately, each with its own rhythm, but this is possibly due to the degenerative changes found at that time in the culture.

When the heart of an embryo 2 days old, in which the atrium and the ventricle are already recognizable, is explanted in vitro, it continues to contract rhythmically at a rate of about one pulsation per second. The contraction starts in the atrium and propagates rapidly toward the ventricle. The electrical records taken immediately after the explantation are composed of two waves only, a quick QRS-wave and a slow T-wave.

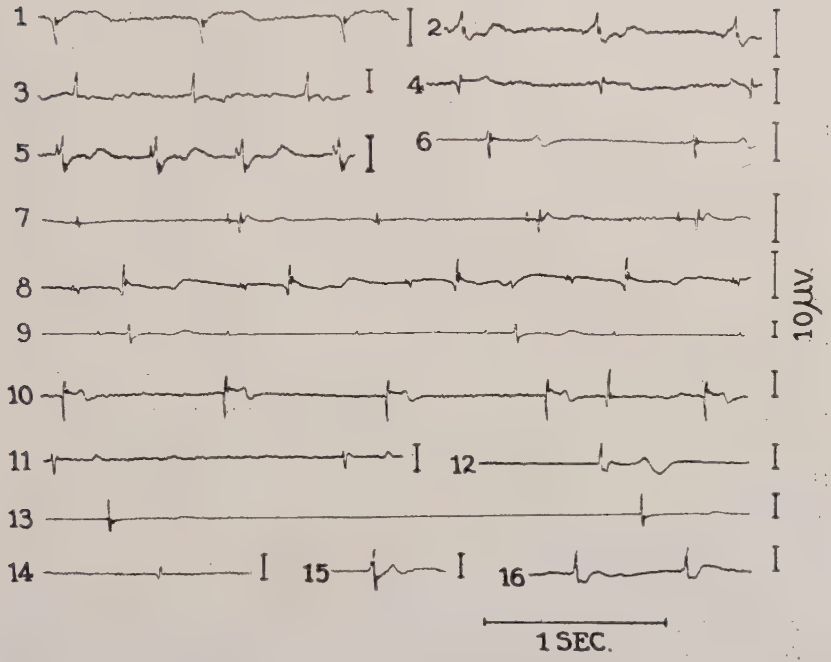


Fig. 1 Electrical records taken with the Grass amplifier from cultures of the heart of chick embryos cultivated in vitro.

1 The heart of a chick embryo 2 days old, record taken immediately after its explantation.
2 Id. taken after 2 days of growth in vitro.
3 The heart of a chick embryo 60 hours old, 2 hours after explantation.
4 One-half of the heart of an embryo 60 hours old, after 2 hours of growth in vitro.
5 The heart of a chick embryo 3 days old, 2 hours after its explantation in vitro.
6 One-third of the heart of a chick embryo 3 days old, after 1 day of growth in vitro.
7 One-half of the heart of an embryo 3 days old, after 1 day of culture.
8 A fragment of the heart of an embryo 4 days old, after 3 days of growth in vitro.
9 A fragment of the heart of an embryo 5 days old, after 3 days of growth in vitro.
10 A fragment of the heart of an embryo 12 days old, after 1 day of growth in vitro.
11 The ventricle of an embryo 2 days old, after 1 day of growth in vitro.
12 The ventricle of an embryo 4 days old, after 1 day of growth in vitro.
13 One-half of the ventricle of an embryo 3 days old, after 1 day of growth in vitro.
14 The atrium of an embryo 2 days old, after 7 days of growth in vitro.
15 The atrium of an embryo 3 days old, after 7 days of culture.
16 The atrium of an embryo 4 days old, after 1 day of culture.

After 1 or 2 days of growth in vitro the same explant gives a complete electrocardiogram with all the three characteristic waves. The P-wave, which did not exist in the former records, becomes more and more apparent with the days of culture. It precedes the QRS-wave at a regular interval of about 50 milliseconds.

In the case of older embryos (from 60 hours to 3 or 4 days of incubation) a complete electrocardiogram, with all the three waves — P, QRS, and T — was obtained from the heart immediately after its explantation. After one or several days of growth in vitro, there was an increase only in the size of the different waves, but the tracing as a whole remained the same, unless degenerative changes made their appearance in the culture. In this case, a complete or incomplete blockage and an auriculo-ventricular dissociation, with a rhythm of two or three to one, appeared.

The hearts of embryos from 60 hours to 3 days old, were divided longitudinally into two or three parts, each containing a fragment of the atrium and of the ventricle, and explanted separately in vitro. The records of each of these explants were analagous to those of an entire heart; all three waves — P, QRS, and T — were present, but reduced in size. Very often, of the two or three cultures obtained by the longitudinal division of the heart of an embryo 3 days old, only one contracted normally, while in the others the electrocardiogram revealed an auriculo-ventricular dissociation. In these cases the auricular rate of pulsation was normal, or about one pulsation per second, while the ventricle contracted only once every 2 or 3 seconds. The same results were obtained from explants of the heart of older embryos (4 days to hatching) containing both auricular and ventricular tissue. The electrocardiogram of only some of them is normal, while in the others a complete or incomplete auriculo-ventricular dissociation was observed. Moreover, it was found that the frequency of the ventricular contraction decreases gradually with the age of the embryo, while that of certain portions of the atrium remains almost unchanged. A rhythm of one to two or one to

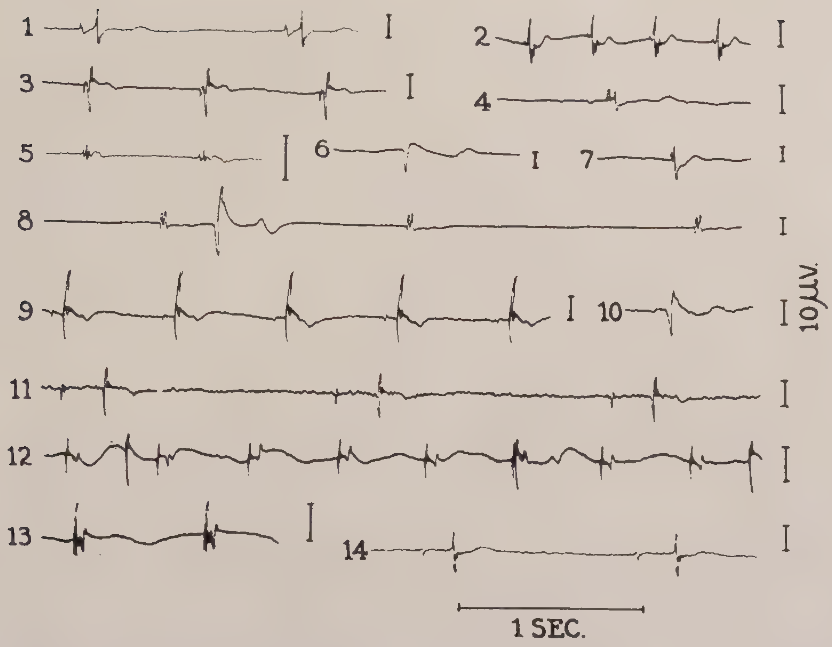


Fig. 2 Electrical records taken with the Grass amplifier from cultures of the heart of chick embryos cultivated in vitro.

1 Atrium of an embryo 5 days old, after 3 days of growth in vitro.
2 A fragment of the atrium of an embryo 10 days old, after 2 days of growth in vitro.
3 A fragment of the atrium of an embryo 14 days old, after 2 days of growth in vitro.
4 A fragment of the atrium of a rat embryo 12 days old, after 4 hours of growth in vitro.
5 A fragment of the atrium of a rat embryo 12 days old, after 6 days of growth in vitro.
6 The ventricle of a chick embryo 4 days old, after 1 day of growth in vitro.
7 The atrium of an embryo 4 days old, after 1 day of growth in vitro.
8 A tracing of the two former together.
9 The atrium of a chick embryo 5 days old, after 1 day of growth in vitro.
10 The ventricle of a chick embryo 5 days old, after 1 day of growth in vitro.
11 A tracing of the two former combined.
12 Fragments of the atrium and of the ventricle of a chick embryo 11 days old, after 1 day of culture (the contact between the two fragments was not complete).
13 The atrium of the former preparation after the ventricle was removed.
14 Fragments of the atrium and of the ventricle of a rat embryo 18 days old, after 8 days of growth in vitro. The two fragments fused and gave a normal electrocardiogram.

three was observed in explants containing a fragment of the atrium and of the ventricle of embryos 4 to 5 days old, and one to six or one to ten in explants from embryos 12 to 15 days old.

In another series of experiments auricular and ventricular tissues were cultivated separately with the following results. The ventricle of a chick embryo of 48 to 60 hours old contracts in tissue culture from the moment of its explantation. Its frequency, however, is half of that of an entire heart, and on the electrical records, taken from this culture immediately after the explantation or several days later, only the QRS and T waves appear, the P wave being absent.

Fragments of the ventricle of older embryos, from 4 days to hatching, behave in the same way; they give electrical records with only two waves, but their frequency and automatism gradually decrease with the age of the embryo and with the distance of the fragment from the atrium.

The atrium of an embryo 2 days old contracts in tissue culture from the moment of its explantation, at a rate of one pulsation per second. The electrical records, taken at that time, do not show any typical wave; single diphasic waves appear in the records taken from the same explant after 2 or 3 days of growth in vitro.

The electrical records of an atrium from an embryo 3 days old show, from the beginning, two waves — one, quick diphasic, and the other slow, di- or monophasic. These two waves are similar to the QRS and T of the normal electrocardiogram. After 7 days of growth in vitro the same explant gives a more complete electrical tracing with three waves; a small wave analagous to the P-wave precedes the former two waves. The same kind of tracings, with all three waves, were obtained from only small fragments of the atrium of older embryos cultivated in vitro.

The fact that an isolated auricle, or even part of it, gave a complete electrocardiogram with three waves, instead of one with a single small P-wave, raised the question whether normally the ventricle does not have a reducing effect on the

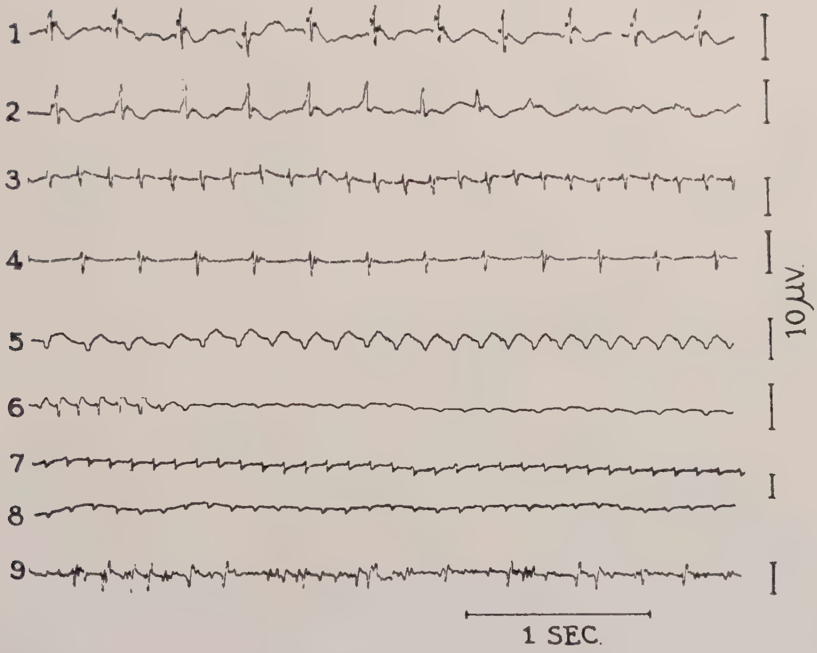


Fig. 3 Electrical records taken with the Grass amplifier from cultures of skeletal muscle cultivated in vitro.

1 and 2 Parts of one record of a skeletal muscle of a chick embryo 9 days old, after 5 days of growth in vitro. The muscle contracted rhythmically. At the end of the series (2) the waves became gradually smaller.

3 A record of a skeletal muscle of a chick embryo 10 days old, after 4 days of growth in vitro.

4 Id. of an embryo 9 days old, after 5 days of growth in vitro.

5 An explant containing more than one muscle, each contracting with a different rhythm. It came from an embryo 9 days old, and was cultivated during 4 days. The waves are of the slow type.

6 Skeletal muscle of a chick embryo 12 days old, after 3 days of growth in vitro. While one muscle was active, waves of a certain amplitude were obtained. The waves became smaller and slower as soon as several muscles started to contract simultaneously, but with a different rhythm.

7 and 8 Parts of a single record of an explant of a chick embryo 10 days old, after 4 days of growth in vitro. Quick waves of a certain amplitude are produced by the contraction of a single muscle; the small waves which follow them (8) are due to the simultaneous contraction of several different muscles.

9 A record of a fragment of a chick embryo 3 days old, containing three myotomes and the corresponding portion of the spinal cord, after 5 days of growth in vitro. The muscular contractions in this case were neurogen; they were not rhythmic.

action currents of the atrium. In order to solve this problem, two series of experiments were undertaken. In the first series, fragments of the atrium and of the ventricle were cultivated separately and then, after electrical records from each of them were obtained, they were placed, side by side, in the same coagulum and a tracing taken of both of them together. In the second series of experiments the fragments of the atrium and of the ventricle were cultivated from the beginning in the same coagulum and, after an electrocardiogram of both of them was obtained, the ventricle was removed and a tracing from the atrium alone taken.

The results from these two series of experiments were analogous. It was found that the two fragments cultivated in the same coagulum contract each with its own rhythm. They do not seem to influence each other, at least in the beginning. The action currents, however, showed great changes; those of the atrium were significantly reduced. Instead of the three waves, as in the previous experiments, the fragment of the atrium produced only one or two small waves. After the removal of the ventricle from the culture, the same explant of the atrium gave again a record with the three typical waves.

It will be noted that the changes in the electrical tracings just described are marked only when the two fragments are in good contact and the explant of the ventricle is very active. In some of these cultures a complete fusion of the two fragments (from the atrium and the ventricle) took place after 5 to 7 days of growth in vitro, and then a typical electrocardiogram with the three characteristic waves (P, QRS, and T) was obtained.

In summary, the heart tissue possesses, from the beginning, a tendency to organize itself into a single functional unit in which the rate of contraction is determined by the portion of the highest frequency. The differentiation in the heart muscle of a center of higher frequency and of conductive tissue becomes evident at a very early stage. From the last center the frequency decreases gradually to the distal portion of the ventricle.

SOMATIC MUSCLE

As was already pointed out, certain precautions have to be taken in order to obtain normal differentiation and activity of skeletal muscle in tissue culture. The medium has to be poor in embryo juice; when the coagulum is rich in nutritive substances there is an abundant growth of fibroblasts and myoblasts, but there is no differentiation nor contraction of the muscle fibers. Another point which must be taken into consideration is the thickness of the coagulum, since it was found that when this is thin (less than 1 mm) the explant rapidly loses its unity, its elements migrate in every direction, and each myoblast starts to contract separately. In a thick coagulum (close to 2 mm) the cellular migration is reduced and the explant in its entirety becomes active. The size of the explant is also of importance for in order to avoid early degeneration it should be as small as possible.

If these precautions are taken, myotomes of chick embryos from 1 to 3 days, completely isolated from the nervous tissue and cultivated in vitro, differentiate normally and acquire spontaneous activity. At the beginning, 1 or 2 days after the explantation, only isolated myoblasts become active, while 5 to 6 days later the entire explant contracts.

In the case of older embryos (8 to 14 days) explants were prepared from the muscular tissue of the extremities. They generally became active after only 1 to 2 days of growth in vitro. In cultures of somatic muscle from embryos 15 to 17 days old, activity was again delayed and did not appear before 4 or 5 days of growth in vitro. Skeletal muscle of embryos older than 18 days or of new-born chickens seems to have lost its spontaneous activity, and does not start to contract even after 2 months of culture. It was found, however, that when the same muscle is cultivated side by side with an explant of a young embryo (8 to 10 days old) it may again become active. It then maintains its acquired contractility even when it is isolated from the young explant and cultivated separately.

As to the mode of activity of skeletal muscle in tissue culture, it is quite different from that of the myocardium. Like the latter, it contracts rhythmically, but its activity is not continuous; series of contractions are generally followed by rest intervals of various lengths of time. The frequency, the number of contractions in each series, and the length of the rest intervals are always the same for one muscle. They vary, however, from one explant to another. While some muscles have a low frequency, contracting only once every 2 to 4 seconds, others are more active and contract one, two or three times in a second. The same was observed with the rest intervals which are only from 1 to 5 minutes in one muscle, and more than a ½ hour in another muscle.

The difference in rhythm (frequency, number of contractions in each series and the length of the rest intervals), which distinguish one muscle from another, was observed not only when these muscles were cultivated separately, but even when they were explanted in the same coagulum. In a culture containing two or three myotomes (explants from embryos 2 days old) or the anlage of two or three different muscles (explants from older embryos), each of the differentiating muscles contracted with its own rhythm, without being influenced by its neighbor, even after 2 months of growth in vitro.

The same was observed with isolated myoblasts or muscle fibers which, as was pointed out previously, start to contract almost immediately after the beginning of the culture, before the entire explant becomes active. Each of these muscle cells contract with its own rhythm. Even later, when the explant is already active in its entirety, young myoblasts are continuously appearing in the zone of growth where their study is relatively easy, due to the thinness of the preparation at that level. These myoblasts start to contract each with its own rhythm, independently of the rest of the explant, before the appearance in them of any signs of differentiation. Myofibrillae and cross-striation could not be detected in these myoblasts before 1 or 2 days of activity.

Using the same technic as for the explants of the myocardium, an attempt was made to register the electrical activity of skeletal muscle in tissue culture. A great number of explants were used in these experiments but only in a few cases good records were obtained. One of the difficulties was the presence in a great number of the cultures, used for this experiment, of several foci of activity. It was found that when in a culture there are fragments of different muscles contracting simultaneously, but each with a different rhythm, the electrical record is either a straight line, or composed of waves of a very low potential.

Good records, with waves well defined, were obtained from explants in which all the muscle fibers had the same frequency and contracted simultaneously. Such cultures, however, were hard to obtain because of the fact that the explants were mostly isolated from young embryos in which the limit between the different muscles is not yet well defined.

The second difficulty was the fibrillar twitching which was observed, generally in explants from older embryos, at the opening of the preparation. For this reason cultures from young embryos were used for the action current study, although fibrillar contractions could not completely be avoided. In the last case the abnormal activity was observed only after a long exposure of the culture. If at the moment the preparation is opened, and connected with the electrodes, it is in its rest interval, and does not start to contract during the first 5 or 10 minutes, the drying of the coagulum causes the beginning of fibrillar contractions and consequently the loss of the explant for the study of its electrical activity.

No action currents could be obtained from single contracting muscle fibers, neither could it be determined whether the negative results in this case were due to presence in the same culture of other contracting fibers or to the smallness of the current generated by a single cell. Action potentials of a certain voltage were obtained from explants rather small, but which constituted a single functional unit. These action potentials, as seen from the records, are single bipolar waves of the duration of about 50 milliseconds, the voltage varying from one explant to another. The wave is of the rapid type and it is not preceded nor followed by secondary waves. Generally, the action potentials of a series of contractions are all of the same voltage. Sometimes, however, at the end of the active period the waves become gradually smaller and slower.

When, besides the main contracting muscle in the culture, there are one or more small secondary foci of activity, instead of rapid bipolar waves, slow monopolar waves of a low voltage are obtained. One explant may give a record with these two forms of waves. In such cases it was established, by microscopical observation, that while the main muscle of the culture alone was active, quick bipolar waves were produced. These were replaced by slow waves of a low voltage whenever the secondary foci contracted simultaneously with the main muscle, but with a different rhythm.

A complete account of the electrical activity of innervated skeletal muscle will be given in due time in another paper. At present, it must be emphasized that when myotomes of a chick embryo of 1 to 3 days old are explanted in vitro, together with the corresponding fragment of the spinal cord, they continue their differentiation normally and start to contract at the same time as during normal development, or at the fourth day of life. The contractions in this case are neurogenic, due to the activity of the nerve cells of the fragments of spinal cord present in the culture. They are not rhythmic, as can be seen from their electrical records.

The difference between the activity of the innervated skeletal muscle and the non-innervated is consequently in the rhythm (it is rhythmic in the last and arhythmic in the former), and in the moment of initiation of the contractions. The contraction of isolated myoblasts starts in both at a very early stage, but the activity of the explant in its entirety is earlier in the presence of nerve cells.

The electrical records of the innervated muscle are very complicated; they show a great number of waves of various size and frequency. The nature of these waves, whether they are due to the activity of the nerve cells or to the contraction of the muscle could not yet be determined, consequently it will not be discussed in the present paper.

DISCUSSION

The contractibility of the skeletal, as well as the heart muscle fibers, seems to precede their morphological differentiation, and from this point of view there is very little difference between these two types of muscle. Goss ('33–'38) studied in tissue culture the development of the heart in rat embryos and found that it starts to pulsate at the stage of three somites in the complete absence of striations or other signs of cellular differentiation. Copenhaver ('39) came to the same conclusion in his studies on Amblystoma punctatum embryos in which he could not detect any myofibrillae nor cross-striation at the moment the heart begins to contract.

In the case of skeletal muscle, Wintrebert ('05–'20) is the only one who observed the contraction of the myotomes in embryos of Selachian before the heart started to pulsate. His observation did not find any confirmation in the work of other investigators. According to Coghill ('29), Windle ('31–'34) and others, who studied the behavior of embryos of different species of animals, the somatic muscle becomes active at a relatively advanced stage, or at least not before it receives its innervation.

From observations made in tissue culture it seems, however, that the skeletal muscle is quite precocious in its activity. Lewis ('20), as was mentioned above, observed the contraction of isolated myoblasts in cultures of skeletal muscle of chick embryos 4 to 10 days old. The present study seems to confirm this observation. Moreover, it was found that at the moment the myoblasts start to contract, they are devoid of myofibrillae. It is true that the explant, as a whole, does not become active before several days of growth in vitro, but the contraction of isolated myoblasts is observed almost from the moment of the explantation. Later, when the explant is already active and advanced in its differentiation, young myoblasts are still found in the zone of growth, where, as it was stated above, their differentiation into cross-striated muscle fibers does not precede their activity, but follows it.

If, as was just stated, the skeletal muscle cells become contractile at the same stage as those of the myocardium, namely at the stage of undifferentiated myoblasts, the question then arises as to difference in behavior of these two kinds of tissue during normal development: Why does the heart start to contract in a chick embryo as early as 33–35 hours of incubation, while the skeletal muscle does not exhibit any activity before the age of 88 to 90 hours (Windle, '31; Kuo Zing Yang, '32)? Is it because of the presence of unity in the former and its absence in the latter?

In the case of the myocardium, even when it is transplanted in vitro, there is no tendency of isolated myoblasts to contract separately; the entire explant becomes active at once. This is possibly due, at least in part, to the syncytial structure of this organ. Another factor which seems to be of importance in the functional unity of the heart, is the gradual decrease in automatism and frequency in the part of the myocardium distal to the venous sinus, and the subordination of segments of lower frequency to those of higher frequency.

The difference in frequency between the auricular and ventricular musculature becomes apparent in embryos 2 days old, and although no electrical activity could be registered from the atrium at that time, its dominant influence over the ventricle is already evident. In its absence, the rate of contraction of the latter is almost half of that of the entire heart.

The gradual decrease in frequency in the musculature of the ventricle becomes more and more apparent with the development of the embryo. It is about one-half to one-third of that of the atrium in embryos 3 days old, and only one-sixth to one-tenth in embryos 15 to 20 days old.

It is true that in explants of the myocardium, functional unity does not always exist; very often the fragments of the atrium and of the ventricle contract each with its own rhythm, in spite of being in contiguity or even in continuity one with the other. But, as was pointed out, in the case of the combined cultures, the unity may finally be achieved if the culture is kept alive long enough for the two fragments to become completely fused. In this case, again, the fragment of the ventricle with a lower frequency falls under the influence of that of the atrium which is the most active.

In the case of the skeletal muscle there is a tendency toward functional unity, but not of the same nature as in the myocardium. There is no difference in frequency from one fiber to another in the same muscle. Indeed, the functional unity of a muscle seems to be due to the fact that all the fibers which enter in its constitution are of the same frequency and contract simultaneously. This, (the simultaneous contraction of all the fibers of a single muscle) however, appears relatively late in the embryo as well as in tissue culture. At the beginning, as was pointed out, only isolated myoblasts are active; they do not have any influence upon the rest of the explant because, possibly, of the absence of anastomosis among the skeletal muscle fibers, as in the case of the myocardium.

The lack of unity, at the beginning, among the fibers of a single muscle, and the contraction of isolated myoblasts, cannot be explained on the basis of the absence of anastomosis, since it was shown that there is a difference in behavior between explants containing a fragment of the spinal cord and those without any nervous tissue. In the former the activity of the entire muscle starts 3 to 4 days earlier than in the latter, in spite of the fact that structurally the two kinds of explants are analogous. The spinal cord does not seem to have any influence upon the differentiation of the skeletal muscle, this takes place in the explants in the complete absence of nervous tissue. The earlier appearance of activity in the presence of a fragment of the spinal cord may be due to its coordinating action upon the different myoblasts of each muscle, or to a more precise distribution of a chemical substance involved in the muscular activity.

The spontaneous activity of skeletal muscle in tissue culture must be due to a chemical substance produced by the same tissue, otherwise it is very hard to understand its mechanism. In favor of this hypothesis is also the fact that muscle of older embryos, or of a new-born chicken, recover their lost activity when they are cultivated together with an explant from a young embryo. The induction of activity, in this case, cannot be explained on the basis of a coordinating effect of the young muscle, because if this were the case, both explants would contract with the same rhythm. The fact that each of these muscles contract with a different rhythm, and that the older explant, once it has started to contract, continues to be active, even after its isolation from the young muscle and its cultivation separately, is in favor of the above hypothesis.

Admitting that the activity of somatic muscle in tissue culture is due to a chemical substance which is produced by the muscular tissue itself, or by the nerve endings, in explants of myotomes with fragments of the spinal cord, the difference in behavior of these two kinds of explants may be explained as follows: The moment of initiation of activity in innervated muscle is precocious due, possibly, to the greater amount of the stimulating substance produced by the nervous tissue, and to its direct action on the muscle fibers at the level of the nerve endings. In the case of non-innervated skeletal muscle, the substance produced by this tissue must act by diffusion, and unless it is produced in great amounts, it will not cause the contraction of the entire explant. The threshold which decreases with the muscular differentiation may also play an important part in this case.

The substance involved in the spontaneous activity of skeletal muscle in tissue culture seems to be acetylcholine-like (Szepsenwol, '42; Szepsenwol and Caretti, '42; Sacerdote de Lustig, '42). It is consequently different from that of the myocardium in which the automatism is due to an adrenalinlike substance, as was established by Haberlandt ('26) and others. This basic difference between the skeletal and cardiac muscle can be demonstrated in tissue culture, where the former seems to have an inhibitory effect upon the latter, its effect being neutralized by atropine (Szepsenwol, '42).

In addition to the differences just mentioned, the spontaneous activity of skeletal muscle can be distinguished from that of the heart muscle by its action currents. Multiple waves were obtained from the entire heart or from fragments of it, while from explants of skeletal muscle the action currents appear in the form of single bipolar waves. This difference in the action currents may be due to the structural differences of these two types of muscle. A skeletal muscle is formed of fibers of the same frequency; its action currents will consequently be the sum of the electrical activity of all its fibers contracting simultaneously. In the case of the myocardium, which is formed from elements of different frequencies, the action currents are possibly the sum of the electrical activity of fibers contracting not simultaneously, but successively.

In the case of explants of skeletal muscle, it was shown that when several muscles of different frequencies contract simultaneously, each with its own rhythm, the rapid bipolar wave is replaced by a slow monopolar wave of a low voltage. In view of this fact, one wonders whether the slow T-wave of the electrocardiogram is not due to an analogous phenomenon, whether it does not represent the sum of electrical activity of fibers of different frequencies contracting simultaneously at the end of each cardiac cycle.

SUMMARY

Skeletal and cardiac muscle of chick embryos have been cultivated in vitro and their activity in relation to their morphological differentiation studied. It was found that in both types of muscle, activity precedes the appearance of myofibrillae and of cross-striation. The difference in behavior of heart and skeletal muscles seems to be due in part to the difference in organization of these two types of muscle.

The heart is formed of a syncytium of fibers in which the automatic power and the frequency of contraction decreases gradually from the atrium to the distal extremity of the ventricle. The functional unity of the heart, or of a fragment of it in tissue culture, seems to be due to this peculiar arrangement of its muscle fibers, in which the cells with less automatic power and of lower frequency are dominated by those of greater automatism and higher frequency. When isolated fragments of the atrium and of the ventricle are cultivated side by side, they contract at the beginning each with its own rhythm, but later, when fusion between them is accomplished, they form a single contracting unit with a frequency of that of the atrium.

The functional unity in a skeletal muscle seems to be based on a different principle, namely, on its constitution of fibers of the same frequency of contraction. When fibers of different frequencies are explanted in the same culture they conserve their own rhythm, without being dominated by cells of higher frequencies, even after 2 months of growth in vitro.

A difference in the action currents of the explants of the two types of muscle was found. Multiple waves were obtained from explant of the myocardium, and a single bipolar wave from those of skeletal muscle. This difference seems to be due to the fact that in the skeletal muscle all the fibers are of the same frequency and are contracting simultaneously, while in the myocardium they are of different frequencies and are contracting in succession.

It was found that when in a culture there are several different muscles contracting simultaneously, but each with its own rhythm, the quick bipolar wave is replaced by a slow mono- or bipolar wave of a low voltage. On the basis of this observation it was suggested that the slow T-wave of the electrocardiogram may be due to the simultaneous contraction of muscle fibers of different frequencies, following the impulse received from the pace-maker.

In the automatism of the heart an adrenaline-like substance seems to be involved (Habderlandt, '26), and in this respect it also differs from the spontaneous activity of the skeletal muscle which seems to be due to an acetylcholine-like substance.

LITERATURE CITED

COPENHAVER, W. M. 1939 Initiation of beat and intrinsic contraction rates in the different parts of the Amblystoma heart. J. Exp. Zool., vol. 80, pp. 192–224.

COGHILL, G. E. 1929 Anatomy and the problem of behavior. Cambridge.

GOSS, C. M. 1933 Observations on the differentiation of cardiac muscle in tissue culture. Arch. Exp. Zellforsch., vol. 14, pp. 175–201.

——— 1937 Early development of the rat heart in vitro. Anat. Rec., vol. 67, p. 20.

——— 1938 The first contractions of the heart in rat embryos. Anat. Rec., vol. 70, pp. 503–524.

HABERLANDT, L. 1926 Ueber ein Hormon der Herzewegung. Weitere versuche mit Froshherzextrakten. Pflug. Arch., vol. 214, p. 470.

HOGUE, M. J., AND G. S. RENYI 1939 Giant muscle cells in tissue culture. Arch. Exp. Zellforsch., vol. 23, p. 122.

KUO ZING YANG 1932 Ontogeny of embryonic behavior in Aves. I. The chronology and general nature of the behavior of the chick embryo. J. Exp. Zool., vol. 61, p. 395; ;vol. 62, p. 453.

LEWIS, M. R. 1920 Muscular contraction in tissue culture. Contr. Embryol. Carneg. Inst., vol. 9, p. 121–212.

SZEPSENWOL, J. 1941 Las primeras contracciones embrionares en cultivos in vitro. Rev. de la Soc. Argentina de Biol., vol. 17, pp. 336–347.

——— 1941 Contracciones espontaneas de musculo somatico de embriones de Pollo en cultivos in vitro. Rev. de la Soc. Argentina de Biol., vol. 17, pp. 491–498.

——— 1942 Las modificaciones del automatismo muscular en el desarollo embrionario estudiado en cultivos in vitro. Rev. de la Soc. Argentina de Biol., vol. 18, pp. 71–78.

——— 1942 Inducción del automatismo contractil en musculos de Pollito cultivados in vitro. Rev. de la Soc. Argentina de Biol., vol. 18, pp. 294–299.

——— 1942 Cultivos simbioticos de musculo somatico, liso intestinal y cardiaco de embriones de pollo. Rev. de la Soc. Argentina de Biol., vol. 18, pp 517–523.

SZEPSENWOL, J., AND J. A. CARETTI 1942 Acetilcolina en embriones de pollo. Sus variaciones en los musculos estriados y en el encefalo durante el desarollo. Rev. de la Soc. Argentina de Biol., vol. 18, pp. 300–307.

——— 1942 Acetilcolina en embriones de pollo. 2, Sus variaciones en los differentes centros nerviosos durante el desarollo. Rev. de la Soc. Argentina de Biol., vol. 18, pp. 532–538.

SZEPSENWOL, J., AND J. B. ODORIZ 1943 Potenciales de accion del esbozo cardiaco de embriones de pollo y de rata cultivados in vitro. Rev. de la Soc. Argentina de Biol., vol. 19, pp. 279–295.

SACERDOTE DE LUSTIG, E. 1942 Estudio del automatismo muscular en cultivos in vitreon acetilcolina, eserina, adrenalina y atropina. Rev. de la Soc. Argentina de Biol., vol. 18, pp. 523–537.

WINDLE, W. F. 1931 The neurofibrillar structure of the spinal cord of cat embryos correlated with the appearance of early somatic movements. J. Comp. Neurol., vol. 53, pp. 71–114.

———— 1936 Genesis of somatic behavior in mammals. J. Physiol., vol. 87, pp. 31–33.

WINDLE, W. F., J. E. O'DONNELL AND E. E. GLASSHAGLE 1933 The early development of spontaneous and reflex behavior in cat embryos and foetuses. Physiol. Zool., vol. 6, pp. 521–541.

WINDLE, W. F., AND D. W. ORR 1934 The development of behavior in chick embryos: spinal cord structure correlated with early somatic activity. J. Comp. Neurol., vol. 60, pp. 287–307.

WINDLE, W. F., W. L. MINEAR, M. R. AUSTIN AND D. W. ORR 1935 The origin and early development of somatic behavior in the albino rat. Physiol. Zool., vol. 8, pp. 156–185.

WINTREBERT, P. 1920 La valeur comparée et le déterminisme des signes principaux de la contraction myotonic aneurale observée chez les embryos de sélaciens. C. R. Acad. Sci., Paris, vol. 171, pp. 1086–1089.

THE DAY TO DAY LEVEL OF ESTROGEN AND PROGESTIN DURING LACTATION IN THE MOUSE[1]

WILLIAM B. ATKINSON AND JAMES H. LEATHEM[2]
Department of Anatomy, College of Physicians and Surgeons, Columbia University, New York, and Department of Zoology, Rutgers University, New Brunswick, New Jersey

ONE TEXT FIGURE AND ONE PLATE (SIX FIGURES)

The production of the ovarian hormones during pregnancy and pseudopregnancy in the mouse has been reported in an earlier paper (Atkinson and Hooker, '45). The criteria of hormonal stimulation used in that study were the endometrial changes specifically elicited by estrogen and progesterone (Hooker, '45). The present study is an extension of these observations to the period of lactation in the mouse.

MATERIALS AND METHODS

Fifty-seven young adult female mice were used. In order to minimize individual variation, all were members of the Swiss albino strain. The animals were mated and then isolated in individual cages. They were examined every morning to determine the date of parturition. The young in our colony are generally weaned on the twenty-first or twenty-second day post partum, therefore the latter day has been considered the termination of lactation. Again to minimize individual variation, the size of the litter was limited to six animals on the second day post partum. The nursing mothers were then divided into two groups for subsequent observations.

---

[1] Aided by a grant, administered by Dr. Philip E. Smith, from the Rockefeller Foundation, New York.

[2] The authors wish to express their appreciation of the aid given by Dr. Charles W. Hooker in the interpretation of the present material.

In the first group a total of thirty-four animals was killed at daily intervals from parturition to the twenty-first post partum. The animals were killed with illuminating gas, and the entire genital tract was removed and placed in Bouin's fixative. A segment from the middle portion of one uterine horn from each specimen was imbedded in paraffin. Sections were cut 5 to 8 micra in thickness and were stained with Ehrlich's hematoxylin and eosin.

The histological changes in the endometrium evoked by administration of estrogen and progesterone (Hooker, '45) were used to identify the action of ovarian estrogen and progestin during laction and to estimate the fluctuations in the level of each hormone. Stimulation by estrogen is identified by the presence of a tall granular uterine epithelium, edema, and leucocytic infiltration of the endometrium. Progesterone stimulation is indicated by the presence of enlarged vesicular stromal nuclei. The effect of each hormone is specific and may be elicited either separately from or concurrently with that of the other. The extent of each morphological pattern is roughly proportional to the level of that hormone.

The second group of twenty-three mice was used to confirm and extend the observations on the vaginal smear and the suppression of estrus during lactation previously reported (Long and Evans, '22; Parkes, '26). The vaginal smear of each mouse in this series was made daily from the second day post partum until the termination of the particular experimental procedure to which the animal was subjected.

OBSERVATIONS

1. *The endometrium during lactation*

On the day of parturition (one animal) the uterine epithelium is very tall and markedly folded. The cytoplasm of the cells is eosinophilic and granular. These are numerous leucocytes in the stroma and uterine lumen. The stroma is slightly edematous, and the stromal nuclei are predominantly fusiform and dense. These morphological features indicate that there is a moderately high level of estrogen and a very low level of progestin acting on the endometrium.

On the first day post partum (one animal) the histological structure of the endometrium is essentially the same as that described on the day of parturition (fig. 4). Areas of degeneration in the epithelium and the disappearance of the leucocytes suggest, however, that the level of estrogen is declining.

On the following day (two animals) the epithelium is low and the mucosal folds have almost disappeared. There is little or no evidence of edema. In one of the animals the boundaries of the epithelial cells are indistinct and the cytoplasm is agranular. About one-third of the stromal nuclei are round, and a few are vesicular. In the other animal the epithelium shows signs of general disintegration. The stromal nuclei are predominantly rounded and approximately one-half are vesicular. The level of estrogen is apparently very low in both animals. The first specimen shows a slight stimulation by progestin and, the second specimen exhibits a very distinct response to progestin.

On the third day post partum (one animal) the general morphological picture approximates that of the second animal described above. However, the stromal nuclei are round and one-third are vesicular, indicating a moderate progestin activity.

On the following day (two animals) the epithelium remains low and the uterine lumen is slit-like. In one specimen there is considerable variation in the condition of the epithelium. In some areas the cells are normal in appearance, in other areas there is marked degeneration. In the second specimen the epithelial cell walls are indistinct, the cytoplasm is eosinophilic and agranular. The stromal nuclei in both animals are round. In the first less than one-half of the nuclei are vesicular, in the second slightly more than one-half are vesicular. Thus, the level of progestin is moderate in the former, and somewhat higher in the latter. Neither specimen shows evidence of estrogen stimulation.

From the fifth through the twelfth days (fourteen animals) the histological picture, although varying somewhat from animal to animal, remains relatively constant (figs. 5 and 6). The epithelium is cuboidal to low columnar in height. The cytoplasm is clear except for an eosinophilic region along the free border of the cells. No edema or leucocytes are present in the stroma. These observations indicate a continued low level of estrogen. The stromal nuclei are rounded and in twelve of the fourteen animals one-half or more are vesicular. During this period, then, there is evidence of continued moderate to high levels of progestin activity.

From the thirteenth through the nineteenth days (ten animals) the epithelium is somewhat taller than during the preceding 7 days. The cytoplasm remains clear except for the eosinophilic border described above. There is no edema or leucocytic infiltration of the stroma. These morphological features are indicative of a continued low level of estrogen. The condition of the stromal nuclei varies from fusiform and dense to rounded and vesicular indicating varying degrees of progestin stimulation. Four of the animals show a marked progestin response, three a slight response and in three there is no indication of progestin stimulation. The over-all impression of this period is that it is one of declining luteal activity.

On the twentieth and twenty-first days (three animals) there is no change from the preceding period (fig. 7) in the epithelium. Stromal edema and leucocytic infiltration are absent. Thus, the level of estrogen remains low. In two of the animals the stromal nuclei are predominantly fusiform and dense, in the third animal there are scattered areas of rounded vesicular nuclei. These is no evidence of progestin stimulation in the first two animals, but a slight indication of a response in the third.

The day to day level of estrogen and progestin as indicated by the endometrial morphology in the lactating mouse may be summarized as follows (fig. 1): On the day of parturition there is evidence of a moderate level of estrogen and a very low level of progestin secretion. Within 2 days estrogen apparently disappears and is not present again during the remainder of lactation. Evidence of progestin secretion increases during the first 4 days post partum to reach a high level on the fifth day. Progestin persists at a moderate to high level until the twelfth day. Thereafter it decreases steadily to reach the initial low level by the twenty-first day when lactation ends.

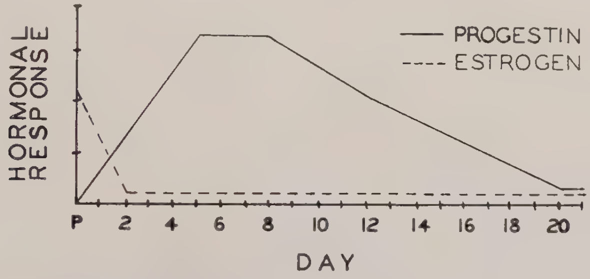


Fig. 1 The sequence and fluctuation in the intensity of the response of the endometrium to estrogen and progestin during the lactation in the mouse. Although intensity is estimated on a comparative basis, no quantitative measurements were attempted. P-day of parturition.

2. *The vaginal smear during lactation*

In a preliminary study the vaginal smears were followed daily on six lactating mice from the second to the twenty-second day post partum. All the animals but one exhibited diestrous smears throughout the entire period. The remaining animal had a smear containing cornified cells on the twenty-first and twenty-second days.

In order to determine the effect of the presence of a litter on the vaginal smear after the twenty-second day, six litters were left with the mothers and the vaginal smears were followed daily until estrus recurred. Completely cornified smears appeared from 24 to 28 days post partum (average 26.5 days). Removal of the litters on the twenty-second day from six mothers did not markedly affect the time of the return of estrus. Cornified smears were obtained in this group from 22 to 28 days post partum (average 25.8 days).

When the young were removed from five actively lactating mothers on the tenth day post partum cornified smears appeared from 3 to 6 days later (average 5.0 days).

DISCUSSION

The accuracy and limitations of the morphological criteria which were used in the present study to estimate the levels of estrogen and progestin during lactation have been fully discussed in previous papers (Atkinson and Hooker, '45; Hooker, '45). In brief, the endometrial changes may be considered specific for the hormones envolved. Experimentally, the degree of morphological alteration is roughly proportional to the amount of the hormone injected. The temporal relations are accurate to at least from 24 to 48 hours, the longest time required for the appearance and disappearance of morphological change in response to injected hormone.

The literature concerned with the production of ovarian hormones during lactation is very sparse. The only quantitative data on the urinary excretion of these hormones have been obtained in post-parturient women. Wilson et al. ('39), found that thirty-four of fifty-one cases showed but low to minimal urinary excretion of pregnandiol on the fourth day after delivery. Hain ('40) reported that in her series of parturient women pregnandiol disappeared by the third to the fifth day post partum. It was further noted that urinary excretion of both combined estrone and combined estriol markedly decreased during the same period. In a study of the human endometrium during the amenorrhea of lactation Topkins ('43) found progestational alterations in only 9 of 145 biopsy specimens. These specimens were secured near the first episode of post partum uterine bleeding. No direct studies on ovarian hormone production during lactation are available in other species.

The present observations on the function of the ovaries during lactation in the mouse can be correlated with the findings of other investigators. Parkes ('29) has reported that the endometrium of the lactating mouse becomes sensitive to deciduomata formation on the fourth day post partum. The duration of sensitivity was not determined. Lyon and Allen ('38) have found that the duration of sensitivity in the rat is from the fourth to the seventeenth day of lactation. These observations are of interest as it is known that progesterone is necessary for the formation and maintenance of the deciduomal reaction (Rothchild et al., '40; Hooker, '41; Atkinson, '44). The appearance of progestin, as determined in the present study, coincides with the appearance of uterine sensitivity. Although there is evidence of moderate to high levels of progestin only until the twelfth day post partum, appreciable quantities of progestin are present almost to the termination of lactation.

The data obtained concerning the vaginal smear during lactation are in substantial agreement with those previously reported in the mouse (Parkes, '26) and in the rat (Long and Evans, '22). A continued diestrous vaginal smear and the absence of evidence of estrogen stimulation of the endometrium during lactation are mutually complimentary phenomena. Suppression of cyclic activity in the ovary is obviously due to the stimulus of nursing since removal of the litter in early lactation is followed by the recurrence of estrus.

The disparity between the present observation that there is considerable luteal activity during lactation in the mouse and previous reports indicating the absence of such activity in woman may undoubtedly be explained on the basis of differences in reproductive physiology. In woman the corpus luteum of pregnancy regresses early and is replaced by the placenta as the major source of the steroid sex hormones. Ovulation and subsequent corpus luteum formation generally do not recur until several months after parturition if the child is nursed. On the other hand, in the mouse and rat ovulation and estrus occur within 24 hours post partum. Mating and fertilization take place at this time. In order that implantation and placentation can follow, the presence of functional corpora lutea is necessary. It has been shown that removal of the ovaries is not detrimental to lactation in the mouse (Parkes and Bellerby, '27) and in the rat (Turner and Gomez, '36). From the foregoing facts we may conclude that the ovary has no function in the process of lactation itself but is concerned solely with the initiation and maintenance of the next pregnancy.

SUMMARY

The changes in endometrial morphology have been used as an index of the action of the ovarian hormones during lactation. Estrogenic stimulation is estimated by the presence of a tall granular epithelium, edema, and leucocytic infiltration of the endometrium. Progesterone activity is indicated by the presence of enlarged vesicular stromal nuclei. On the day of parturition there is evidence of a moderate level of estrogen and a very low level of progestin secretion. Within 2 days the evidence of estrogenic stimulation disappears and does not recur during the remainder of lactation. Evidence of progestin secretion increases during the first 4 days post partum and reaches a high level on the fifth day. Progestin apparently persists at a moderate to high level of secretion until the twelfth day. Thereafter it decreases steadily to return to the initial low level by the twenty-first day when lactation terminates. The observation that the vaginal smear remains diestrus in type substantiates the finding that estrogen is not secreted in appreciable amounts during lactation.

LITERATURE CITED

ATKINSON, W. B. 1944 The persistence of deciduomata in the mouse. Anat. Rec., vol. 88, pp. 271–283.

ATKINSON, W. B., AND C. W. HOOKER 1945 The day to day level of estrogen and progestin throughout pregnancy and pseudopregnancy in the mouse. Anat. Rec., vol. 93, pp. 75–95.

HAIN, A. M. 1940 The excretion of estrogen and pregnandiol by pregnant and parturient women. J. Endocrinology, vol. 2, pp. 104–125.

HOOKER, C. W. 1941 Production of deciduomata in ovariectomized mice receiving progesterone. Proc. Soc. Exp. Biol. and Med., vol. 46, pp. 698–700.

——— 1945 A criterion of luteal activity in the mouse. Anat. Rec., vol. 93, pp. 333–347.

LONG, J. A., AND H. M. EVANS 1922 The estrous cycle of the rat. Memoirs Univ. of Calif., vol. 6, pp. 1–148.

LYON, R. A., AND W. M. ALLEN 1938 Duration of sensitivity of the endometrium during lactation in the rat. Am. J. Physiol., vol. 122, pp. 624–626.

PARKES, A. S. 1926 Observations on the estrous cycle of the albino mouse. Proc. Roy. Soc. B., vol. 100, pp. 151–170.

——— 1929 The experimental production of placentoma in the mouse. Proc. Roy. Soc. B., vol. 104, pp. 183–188.

PARKES, A. S., AND C. W. BELLERBY 1927 Studies on the internal secretions of the ovary; 3. The effects of injection of estrin during lactation. J. Physiol., vol. 62, pp. 301–314.

ROTHCHILD, I., R. K. MEYER AND M. A. SPIELMAN 1940 A quantitative study of estrogen-progesterone interaction in the formation of placentomata in the castrate rat. Am. J. Physiol., vol. 128, pp. 213–224.

TOPKINS, P. 1943 Histological appearance of endometrium during lactation amenorrhea and its relationship to ovarian function. Am. J. Obstet. and Gynec., vol. 45, pp. 45–58.

TURNER, C. W., AND E. T. GOMEZ 1936 The effect of ovariectomy upon lactation in the albino rat. Mo. Agric. Exp. Sta. Bul., no. 370.

WILSON, R. B., L. M. RANDALL AND A. E. OSTERBERG 1939 Studies on pregnandiol. Am. J. Obstet. and Gynec., vol. 37, pp. 59–74.

PLATE 1

EXPLANATION OF FIGURES

All illustrations are photographs (× 300) of transverse sections of uteri stained with hematoxylin and eosin.

2 Endometrium of an ovariectomized mouse which received 16.6 μg of estradiol benzoate in 5 days. The endometrium is thick and edematous. The uterine epithelium is tall and granular. Leucocytes are numerous. The stromal nuclei, however, are unchanged. Compare with figure 3.

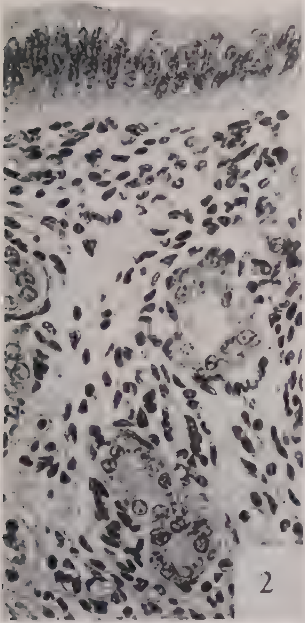
3 Endometrium of an ovariectomized mouse which received five daily injections of 0.25 mg of progesterone. The stromal nuclei are rounded and vesicular. Compare with figure 2.

4 First day post partum. The epithelium is tall and granular. The stroma is edematous, the stromal nuclei are small and dense. These morphological features suggest a moderate level of estrogen is present. There is no evidence of stimulation by progestin.

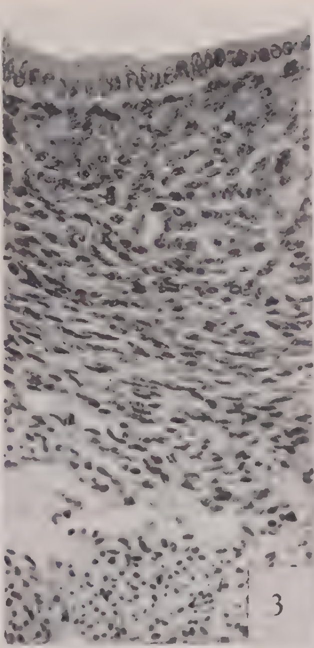
5 Fifth day post partum. The epithelium is low and the cytoplasm is agranular. The stromal nuclei are enlarged and vesicular. A high level of progestin is indicated.

6 Twelfth day post partum. The morphological features are essentially the same as those in figure 5 indicating a continued high level of progestine activity.

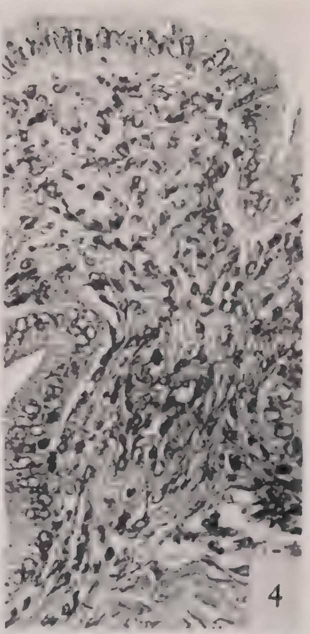
7 Twenty-first day post partum. The epithelium is low, the cytoplasm agranular. The stromal nuclei are fusiform and pyknotic. There is no evidence of stromal edema. The morphological picture is the same as that seen in the castrate, indicating the absence of both estrogen and progestin.



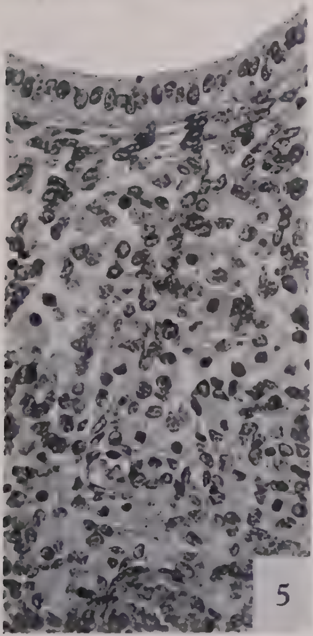
2



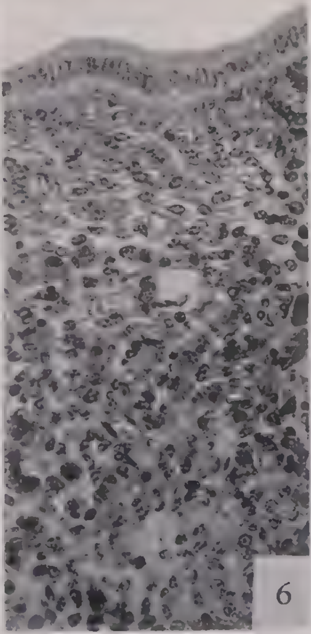
3



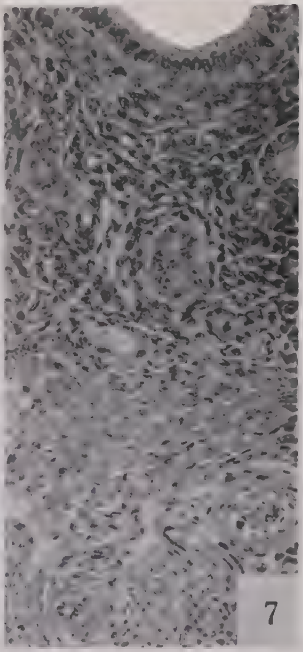
4



5



6



7

THE INTRACELLULAR DISTRIBUTION OF ALKALINE PHOSPHATASE ACTIVITY FOLLOWING VARIOUS METHODS OF HISTOLOGIC FIXATION[1, 2]

VICTOR M. EMMEL

Department of Anatomy, The University of Rochester School of Medicine and Dentistry, Rochester, New York

ONE TEXT FIGURE AND ONE PLATE (SEVEN FIGURES)

Cytochemical studies of the distribution of phosphomonoesterases by the methods of Gomori ('39, '41a, b) have recently disclosed the fact that alkaline phosphatase is regularly associated with the Golgi apparatus of the intestinal epithelium (Emmel, '45) and may be variably present in the Golgi zone of certain other epithelia (Deane and Dempsey, '45). Since knowledge of the intracellular distribution of these and other enzymes must eventually form part of the basis for understanding the activities of the cell as a whole, as emphasized in a recent review (Dempsey and Wislocki, '46), it becomes important to establish, in so far as this is possible, the validity of the results obtained by these methods. In the present study three fundamentally different types of fixation have been employed in the preparation of tissues for the Gomori techniques. These include (1) acetone dehydration, (2) freezing-drying, and (3) fixation in formol-saline.

While this work was in progress Deane and Dempsey ('45) reported, without recording details, that the distribution of

---

[1] The technical assistance for this study was supplied in part by a grant from the Nutrition Foundation to Dr. Karl E. Mason, of this department. Special equipment was obtained through a grant from the Fluid Research Funds of the University of Rochester School of Medicine and Dentistry.

[2] An abstract of this report appears in the Anat. Rec., vol. 94, p. 536 ('46).

alkaline phosphatase in frozen-dried intestine is essentially the same as after acetone dehydration or alcohol fixation. This statement is in general supported by the present observations. After formol-saline fixation the sites of phosphatase activity generally correspond to those demonstrable by the usual techniques. Because of the inactivating effect of formaldehyde, presumably due to reaction with amino groups in the enzyme molecule, this reagent brings to light certain histochemical differences in the alkaline phosphatase of various organs. This is of interest in view of Gould's ('44) finding that formaldehyde is a much stronger inhibitor of kidney phosphatase than of intestinal phosphatase.

MATERIALS AND METHODS

Adult albino mice fed a commercial diet were used in these experiments. After fasting for periods up to 24 hours the animals were killed by decapitation, and tissues were removed as quickly and carefully as possible.

Acetone dehydration. Small specimens were placed in cold (5°C.) acetone for 12 to 18 hours followed by 4 hours in absolute alcohol at room temperature, then cleared in benzene and imbedded in paraffin at 56–58°C. Treatment with alcohol produced sufficient protein denaturation to minimize gross distortion of intracellular structures which often occurs in undenatured specimens during the rehydration and drying which attend the mounting of paraffin sections. By varying the time in alcohol from 1 to 24 hours it was found that the above treatment gave the most uniform structural pictures without observable loss of phosphatase activity.

Freezing-drying. The principles employed were essentially those of previous investigators (Simpson, '41a; Hoerr and Scott, '44). With the iso-pentane commercially available at present it was not possible to attain freezing temperatures as low as have been described. However, it was found that the freezing point of iso-pentane (—158°C.) could be depressed to below —190°C. by adding commercial butane which had been dried by bubbling through concentrated sulfuric acid, thus permitting a more rapid initial freezing of the tissues. Dehydration was carried out in a vacuum system which included a 2-stage oil-diffusion pump giving a final measured pressure of about 8×10^{-7} mm Hg when drying was complete. The temperature during dehydration in different experiments varied from —40 to —78°C. Dehydration required from 1 to 2 weeks depending upon the temperature and the amount of tissue. The dried tissues were treated for 4 hours with absolute alcohol to produce partial denaturation, then cleared in benzene and imbedded in paraffin. The acetone, alcohol, and benzene used throughout these experiments were purified and redistilled. All paraffin sections were cut 5 microns in thickness.

Formol-saline. Merck's neutral reagent formalin (37% HCHO) was used in 10% solution containing 0.9% NaCl. The time of fixation varied from 4 hours to 2 weeks. Frozen sections were cut 10 microns in thickness, washed in distilled water for periods up to 96 hours, incubated individually in the phosphatase substrates, and finally mounted in glycerine jelly.

Phosphatase methods. A. Histochemical. Gomori's ('39) glycerophosphate substrate (pH 9.2 at 37°C.) with the addition of M/100 $MgSO_4$ (Kabat and Furth, '41) was used in these experiments. Paraffin sections were incubated for 3 hours, while frozen sections of the formalin fixed material were incubated for 30 minutes.

B. Quantitative. The effects of formaldehyde on the phosphatase activity of tissue homogenates were determined by the phenolphthalein phosphate method of Huggins and Talalay ('45). M/100 $MgSO_4$ was added to the alkaline substrate because it had previously been incorporated in the histochemical substrate. This increased the activity of the alkaline phosphatase, but produced no fundamental change in the character of the results. In one series of experiments (fig. 1) enzymatic activity was determined in the presence of formalin concentrations varying from 0.001% to 1%. In a second series of experiments (tables 1 and 2) the tissues were fixed in 10%

formal-saline for various times, then homogenized, and the formaldehyde removed from 3 cm³ samples of homogenate by dialysis for 4 hours in cellophane tubing against about 5 gallons of running distilled water at room temperature on a mechanical shaker. Phosphatase activity was determined on 0.5 cm³ aliquots of the various homogenates.

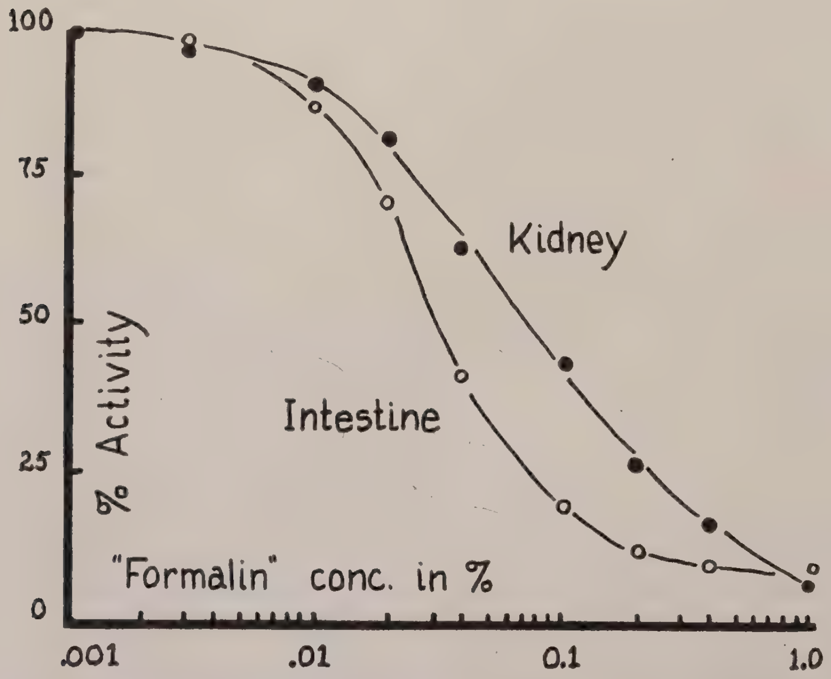


Fig. 1 Graph showing the inhibition of alkaline phosphatase activity in homogenates of kidney and intestine. The formalin concentration present in the final enzyme-substrate mixtures (plotted logarithmically) was attained by adding 0.1 cm³ of an appropriately higher concentration. Activities are plotted in terms of that of an untreated control run at the same time.

OBSERVATIONS

Acetone dehydration. The sites of alkaline phosphatase activity in the nucleus, cytoplasm, cuticular border and Golgi zone of the intestinal cells (fig. 2) have been described in previous publications (Bourne, '43; Emmel, '45; Deane and Dempsey, '45). In a series of specimens taken at intervals of

5 to 10 mm in the upper $\frac{1}{3}$ of the small intestine and at wider intervals in the lower $\frac{2}{3}$, it was found that in the first 3 to 4 cm of the duodenum the cuticular border usually is almost devoid of alkaline phosphatase, while at all lower levels the border regularly shows intense activity. This distribution appears to be independent of the duration of fasting prior to examination.

Freezing-drying. The sites of alkaline phosphatase activity in the intestine (fig. 3) coincide exactly with those demonstrable after acetone dehydration. In cells showing no ice crystals artefacts under oil-immersion examination the preservation of cellular structure is excellent, and in general appearance is superior to that obtained by acetone dehydration. Phosphatase activity in the Golgi zone is generally visualized as a fairly discrete black area with a slightly diffuse boundary; only rarely does it suggest the existence of a network. Activity in the cuticle is prominent at those levels of the intestine where it is regularly found after acetone dehydration. The cytoplasm generally shows slight or moderate activity, with the intensity somewhat greater in the distal half of the cell. Granular foci of phosphatase activity are frequently present in the apical cytoplasm. Nuclei generally show a small amount of alkaline phosphatase activity.

Cellular structure in the kidney is generally better preserved by the freezing-drying technique than by acetone dehydration. After either method phosphatase activity is restricted principally to the brush border and cytoplasm of the proximal convoluted tubules, to nuclei throughout the nephron, and to isolated areas of interstitial connective tissue at the cortico-medullary boundary. These sites of activity have been described and illustrated by Gomori ('41a), Kabat and Furth ('41), Bourne ('43), Menten, Junge, and Green ('44), and Deane and Dempsey ('45). In well preserved specimens the cytoplasm of the proximal tubule cells is so crowded with apico-basally oriented mitochondria that the cells present a striated appearance (fig. 4). In some of these cells alkaline phosphatase activity is clearly associated with the mitochondria, while in others it does not seem feasible to distinguish between diffuse activity in the cytoplasm and that in the mitochondria. Granular foci of alkaline phosphatase activity occasionally occur in cells of the proximal convoluted tubules. The granules are generally distal to the nucleus, but only rarely do their position and structure correspond to that of the Golgi apparatus in these cells. Structures suggesting networks have occasionally been seen in the apical cytoplasm of acetone-dehydrated preparations and much more rarely in frozen-dried material, but these have generally been in areas of the section in which irregular coagulation of the cytoplasm and disruption of the brush border indicate poor preservation of cellular structure and make questionable the interpretation of finer structural details.

Formol-saline. The statement by Gomori ('41b) that formalin inactivates phosphatase has apparently discouraged the use of this reagent in histochemical studies of phosphatase activity. However, alkaline phosphatase can easily be demonstrated in frozen sections of intestine fixed in formol-saline for from 4 hours to 2 weeks. Sections transferred immediately from fixative to substrate show little or no activity, but activity is progressively restored by washing in distilled water up to about 6 hours, following which further washing (4 days) is not attended by further increase in activity. The most prominent sites of activity are in the cuticular border and in the Golgi zone (fig. 5), with scattered granules in the apical cytoplasm. Sections incubated for 1 hour or more show, in addition, weak activity in nuclei and cytoplasm. The Golgi zone in these cells usually appears as a clear "canalicular" structure surrounded by a black deposit of cobalt sulphide. The "canals" are entirely devoid of phosphatase activity, sometimes appear slightly refractile, and can readily be seen in unstained sections. A few pieces of formalin-fixed intestine were passed through acetone and benzene into paraffin. "Canals" surrounded by phosphatase activity can be demonstrated in these specimens (fig. 6).

The inactivation of renal alkaline phosphatase in formol-saline is progressively irreversible until after 3 or 4 days no further activity can be demonstrated. The phosphatase in the brush border is the most resistant to formaldehyde. In specimens fixed for 12 to 24 hours, subsequent washing of the sections restores activity in the brush border and to a lesser extent in the nuclei and cytoplasm of the proximal convoluted tubules (fig. 7). Granular cytoplasmic foci of phosphatase activity are rarely seen in these preparations. Omission of NaCl from the fixative appears to reduce the amount of phosphatase which can subsequently be demonstrated. This effect has been verified in several experiments, but has not been further investigated.

Acid phosphatase can also be demonstrated in frozen sections of formalin-fixed material. The optimal duration of incubation varies from 2 to 24 hours with different tissues. The distribution of this enzyme is somewhat variable after this and the preceding methods of fixation and will not be discussed in detail in the present report. However, it may be stated that renal glomeruli are strongly positive, as well as nuclei in general, and both the cuticular border and to a lesser extent the cytoplasm in the upper prtions of the small intestine. In the rat's spleen (fig. 8) the strong acid phosphatase activity in the red pulp is in marked contrast to the weak activity in the white pulp. The concentric rings of positive cells in the splenic nodules coincide with groups of macrophages capable of absorbing Trypan blue.

Figure 1 records the progressive inhibition of alkaline phosphatase in tissue homogenates by increasing amounts of formalin added to the enzyme-substrate mixtures. From tables 1 and 2 it can be seen that whereas the alkaline phosphatases of intestine and kidney differ markedly in the extent to which activity can be restored by dialysis, this procedure brings to light no such striking differences among the acid phosphatases of liver, spleen, kidney, and intestine. The inhibition of alkaline phosphatase by formalin was also studied histochemically in sections of acetone-dehydrated material incubated in substrate to which formalin had been added in various concentrations. The results were in accord with the quantitative data. Activity in the cuticular border of intestinal cells and the brush border of kidney cells was the last to disappear as the formalin concentration was increased.

TABLE 1

Alkaline phosphatase activity after dialysis of homogenates[1] *of tissue which had been fixed in 10% formol-saline.*

		TIME IN FORMOL-SALINE				
		0 (Control)	2 hr.	2 da.	5 da.	11 da.
% alkaline phosphatase activity	Kidney	100	9	0	..	0
	Intestine	100	23	24	27	16

[1] Homogenates contained about 10 mg wet weight of fresh tissue per cm^3 of final preparation. Activities are calculated in terms of the dialyzed untreated control.

TABLE 2

Effects of formalin on acid phosphatase activity of tissue homogenates.

		"FORMALIN" CONCENTRATION [1] (PER CENT)				TIME IN 10% FORMOL-SALINE [2]	
		0 (Control)	0.4	1.0	5.0	14 hr.	41 hr.
% acid phosphatase activity	Kidney	100	75	45	0	28	17
	Intestine	100	81	50	0	34	27
	Liver	100	68	44	0	27	16
	Spleen	100	89	51	0	31	

[1] Formalin present during incubation of enzyme-substrate mixture. Final dilution attained by adding 0.1 cm^3 of an appropriate concentration of formalin to the substrate.

[2] Tissues fixed in 10% formol-saline, then homogenized and dialyzed. Activities are calculated in terms of that of the untreated, non-dialyzed controls.

DISCUSSION

In those tissues which have been examined by the freezing-drying technique the close correspondence of the distribution of alkaline phosphatase to that seen in chemically fixed specimens strongly supports the general validity of the results obtained by the latter methods. In some preliminary quantitative experiments to determine the effects of various histologic reagents on phosphatase activity it has been found that in tissues passed through acetone, absolute alcohol, and benzene, into paraffin at 58°C., then rehydrated and homogenized, alkaline phosphatase activity is only slightly reduced in comparison with that of homogenized fresh tissues. This further validates the usual histochemical techniques.

The association of alkaline phosphatase with the Golgi apparatus of the intestinal absorbing cells is a remarkably constant feature of the chemical organization of these cells, and is a characteristic which does not appear to be shared generally with other epithelia. Deane and Dempsey ('45) have described alkaline phosphatase in association with the Golgi apparatus in the kidney, liver, and uterus of certain species. However, in the experience of the writer, granules and networks which they have described in kidney and liver have been seen only occasionally even in well preserved specimens; and at present their occurrence, at least in the mouse, does not appear to be sufficiently constant or characteristic to warrant their positive association with the classical Golgi apparatus in these cells. Details of the relationships between this enzyme and intracellular structures will eventually prove important in understanding their roles in the physiology of different cell types; and it is possible that these apparently conflicting observations can be resolved by studies correlating the intracellular distribution of phosphatase with the state of physiological activity of the cells.

The finding of a restricted segment in the first part of the small intestine in which the cuticular border shows little or no alkaline phosphatase activity probably explains the contradictory statements regarding activity in this region of the cell (Bourne, '43; Emmel, '45; Deane and Dempsey, '45).

The significance of the characteristic intracellular distribution of alkaline phosphatase in the intestinal epithelium has not yet been established. It has been suggested (Emmel, '45) that the enzyme in the Golgi zone may play a role in the absorptive functions of these cells, whereas Deane and Dempsey ('45) have favored the view that the enzyme in the Golgi zone of this and other epithelia is destined for secretion rather than a role in intracellular metabolism. In the absence of conclusive experimental evidence, it seems at present inadvisable to argue with great vigor in favor of either hypothesis to the exclusion of the other, since the enzyme may well have both an extracellular and an intracellular role. The term "alkaline phosphatase" probably denotes a large group of phosphatases (Sumner and Somers, '43) all having in common the ability to hydrolyze glycerophosphate; and for this reason the phosphatase in the Golgi zone of certain "nonabsorptive" epithelia may eventually be shown to have properties quite different from that in the intestine. Cytochemical studies employing substrates other than glycerophosphate (e.g., phosphate esters of fatty acids; Lehninger, '46) may provide evidence in this direction; but in the present state of our knowledge it would appear premature to assume that the role of "alkaline phosphatase" is the same in all types of highly differentiated epithelia. Deane and Dempsey have further stated that in the intestine both acid and alkaline phosphatase are most abundant at the base and along the sides rather than at the tips of the villi. In the considerable number of specimens from various levels of the mouse intestine examined by the present writer, this distribution has not seemed as definite as these authors imply. Phosphatase activity in cells at the tips of the villi in acetone-dehydrated specimens (particularly if the intestine has been opened longitudinally prior to fixation) often appears to be as great as elsewhere, and in frozen-dried preparations alkaline phosphatase usually is quite uniformly present in the epithelium throughout the entire length of the villus. Furthermore there seems at present little reason to suppose that the sides of the villi are any less active in absorption than are the tips. Cells at the tips of the villi may well be the oldest and therefore least vigorous (if they arrive there by upward migration from the base, although this is unproven. Andrew and Andrew, '45). Definitive answers to these questions must await the results of carefully planned experiments.

The appearance of "canalicular" structures in the Golgi zone of intestinal cells after formol-saline fixation (figs. 5 and 6) and their absence in acetone-dehydrated or frozen-dried specimens raises the question as to the physical structure of the cytoplasm in this region. The Golgi apparatus has frequently been described as "canalicular" in nature (Hibbard, '45), and in a careful study of the Golgi apparatus by means of the freezing-drying method, Simpson ('41) has described such a canalicular system in the guinea pig duodenum. However, this has not been clearly seen in alkaline phosphatase preparations of frozen-dried mouse doudenum examined in the present study. A detailed investigation of this question was not undertaken, but it might be suggested that the action of formalin may have altered the physical character of materials in the Golgi zone in such a way as to effect a separation or aggregation of constituents which have no alkaline phosphatase activity. The appearance of these "canals" sometimes resembles the myelin forms which have been described and illustrated by Leathes ('25).

It is apparent that Gomori's ('41b) statement that formaldehyde inactivates phosphatase is not strictly applicable under the conditions of the present experiments. The chemical and histochemical effects of formaldehyde here reported are in general accord with the studies by Gould ('44), who found that after dialysis the residual inhibition of phosphatase by formaldehyde was much greater in crude kidney preparations than in a purified preparation of intestinal phosphatase. Gould was inclined to interpret his results as indicating that free amino groups are of lesser importance for the activity of intestinal than for that of kidney phosphatase. From the data here presented (fig. 1) it would appear that in the presence of formalin concentrations of from 0.01% to 1%, alkaline phosphatase activity in whole tissue homogenates of kidney is slightly less inhibited than in similar preparations of intestine. At concentrations greater than

1% both kidney and intestine phosphatase are completely inhibited. A striking difference between kidney and intestine appears after dialysis of material which has been fixed in 10% formol-saline (table 1): alkaline phosphatase activity can be partially restored to the intestine homogenates even after 10 days, while this ability is quickly lost in the kidney homogenates. This probably can be interpreted as a real difference between the renal and intestinal enzymes; but the fact that the two curves of inhibition are so closely parallel (fig. 1) may mean that the difference lies not so much in the relative importance of amino groups in the two enzymes as in the firmness of their union with formaldehyde. It is also possible that changes in physical configuration of the enzyme molecule (Neurath, Greenstein, Putnam, and Erickson, '44) which undoubtedly occur during the denaturation produced by prolonged contact with formalin are of greater significance for enzymatic activity in the case of renal phosphatase. In working with homogenates of whole tissue, one is confronted with all the complexities of the as yet only partially understood interactions between proteins and dilute solutions of formaldehyde (French and Edsall, '45), and it is possible that part of the apparent difference between the renal and the intestinal phosphatases is due to actions of formaldehyde on proteins other than that in the enzyme complex itself. The situation is further complicated by the fact that the crude homogenates probably contain a number of "alkaline" phosphatases (Sumner and Somers, '43). If these differ in their sensitivity to formaldehyde, then any variation in their relative proportions in different tissues would be reflected only as an over-all difference in formaldehyde sensitivity in experiments such as the present.

The existence of a real difference between the alkaline phosphatase of kidney and intestine is further indicated by the earlier work of Bodansky ('37) who found that the former is strongly inhibited by bile salts while the latter is little affected. In cytochemical studies subsequently to be reported (Emmel, '46) it has been found that intestinal phosphatase is strongly inhibited by M/1000 KCN, while renal phosphatase still retains slight activity in the presence of M/100 KCN. The physiological significance of these differences remains to be determined, but the recognition of such differences and their cytological localization must eventually form part of the basis for understanding the functional organization of the cell. At present it seems reasonable to suppose that these differences in respect to phosphatase inhibitors reflect the wide variety of phosphatases and the numerous metabolic processes in which phosphorylation plays a role.

SUMMARY AND CONCLUSIONS

1. The intracellular distribution of alkaline phosphatase has been studied in specimens prepared by acetone-dehydration, the freezing-drying technique, and in frozen sections of material fixed in formol-saline.
2. The general validity of these methods is supported by the fundamental similarity of the results. The first two methods yield practically identical results with conspicuous sites of alkaline phosphatase activity in the cuticular border and Golgi zone of the intestinal epithelium, and in the brush border of the renal proximal tubule. "Granular" foci of phosphatase activity which occasionally occur in cells of the liver and renal proximal tubule, probably do not represent the Golgi apparatus in these cells.
3. Alkaline and acid phosphatase can be demonstrated in frozen sections of material fixed in 10% formol-saline. The alkaline phosphatase distribution is essentially similar to the above, with the exception that the intestinal Golgi zone usually appears as a clear "canal" surrounded by a region of phosphatase activity.
4. By histochemical and quantitative methods the alkaline phosphatases of kidney and intestine were found to differ significantly in the extent to which they are irreverisbly inactivated by prolonged contact with formol-saline.

I wish to thank Mrs. Richard T. Snowman for her expert technical assistance throughout this study.

LITERATURE CITED

ANDREW, W., AND N. V. ANDREW 1945 Mitotic division and degeneration of lymphocytes within the cells of intestinal epithelium in the mouse. Anat. Rec., vol. 93, p. 251.

BODANSKY, O. 1937 Are the phosphatases of bone, kidney, intestine, and serum identical? The use of bile acids in their differentiation J. Biol. Chem., vol. 118, p. 341.

BOURNE, G. 1943 The distribution of alkaline phosphatase in various tissues. Quart. J. Exp. Physiol., vol. 32, p. 1.

DEANE, H. W., AND E. W. DEMPSEY 1945 The localization of phosphatases in the Golgi region of intestinal and other epithelial cells. Anat. Rec., vol. 93, p. 401.

DEMPSEY, E. W., AND G. B. WISLOCKI 1946 Histochemical contributions to physiology. Physiol. Rev., vol. 26, p. 1.

EMMEL, V. M. 1945 Alkaline phosphatase in the Golgi zone of absorbing cells of the small intestine Anat. Rec., vol. 91, p. 39.

——— 1946 A cytochemical study of the effects of KCN on alkaline phosphatase activity in the kidney and intestine. Anat. Rev., vol. 94, p. 459 (Abstract).

FRENCH, D., AND J. T. EDSALL 1945 The reactions of formaldehyde with amino acids and proteins. Advances in Protein Chemistry, vol. 2, p. 277. Academic Press, Inc., New York, N. Y.

GOMORI, G. 1939 Microtechnical demonstration of phosphatase in tissue sections. Proc. Soc. Exp. Biol. and Med., vol. 42, p. 23.

——— 1941a The distribution of phosphatase in normal organs and tissues. J. Cell. and Comp. Physiol., vol. 17, p. 71.

——— 1941b Distribution of acid phosphatase in the tissues under normal and under pathologic conditions. Arch. Path., vol. 32, p. 189.

GOULD, B. S. 1944 The action of "alkaline" phosphatase. J. Biol. Chem., vol. 156, p. 365.

HIBBARD, H. 1945 Current status of our knowledge of the Golgi apparatus in the animal cell. Quart. Rev. Biol., vol. 20, p. 1.

HOERR, N. L., AND G. H. SCOTT 1944 Frozen-dehydration method for histologic fixation. Medical Physics., p. 466. Year Book Publishers, Chicago.

HUGGINS, C., AND P. TALALAY 1945 Sodium phenolphthalein phosphate as a substrate for phosphatase tests. J. Biol. Chem., vol. 159, p. 399.

KABAT, E. A., AND J. FURTH 1941 A histochemical study of the distribution of alkaline phosphatase in various normal and neoplastic tissues. Am. J. Path., vol. 17, p. 303.

LEATHES, J. B. 1925 Role of fats in vital phenomena. Lancet, vol. 208, p. 957.

LEHNINGER, A. L. 1946 The synthesis and properties of the acyl phosphates of some higher fatty acids. J. Biol. Chem., vol. 162, p. 333.

MENTEN, M. L., J. JUNGE AND M. H. GREEN 1944 Distribution of alkaline phosphatase in kidney following the use of histochemical azo dye test. Proc. Soc. Exp. Biol. and Med., vol. 57, p. 82.

NEURATH, H., J. P. GREENSTEIN, F. W. PUTNAM AND J. O. ERICKSON 1944 The chemistry of protein denaturation. Chem. Rev., vol. 34, p. 157.

SIMPSON, W. L 1941a An experimental analysis of the Altmann technique of freezing-drying. Anat. Rec., vol. 80, p. 173.

——— 1941b The application of the Altmann method to the study of the Golgi apparatus. Anat. Rec., vol. 80, p. 329.

SUMNER, J. B., AND G. F. SOMERS 1943 Chemistry and Methods of Enzymes. Academic Press, Inc., New York, N. Y.

PLATE 1

EXPLANATION OF FIGURES

Photomicrographs were taken with apochromatic objectives and compensating ocular on Wratten M plates without a filter. Figures 2 to 6 were taken at an initial magnification of 1000 × and have been reduced to 800 ×. The sites of phosphatase activity are shown as black deposits. No counterstain was applied.

2 Mouse. Small intestine. Acetone dehydration. Alkaline phosphatase is prominent in the cuticular border, Golgi zone, and apical cytoplasm, with lesser activity in the nucleus and basal cytoplasm.

3 Mouse. Small intestine. Frozen-dried. General preservation of cellular structure is superior to that in the preceding specimen, but the distribution of alkaline phosphatase activity is essentially the same.

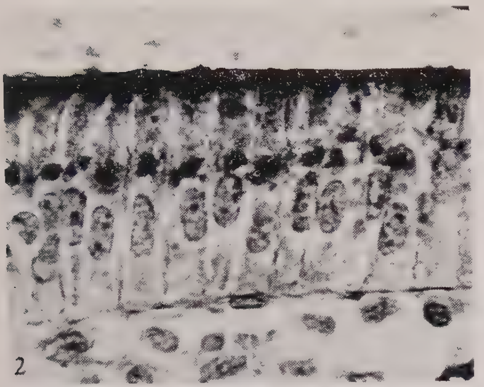
4 Mouse. Renal proximal tubule. Frozen-dried. Alkaline phosphatase activity is prominent in the brush border, and is also present in the nuclei and mitochondria. The parallel arrangement of the latter gives the cells a striated appearance.

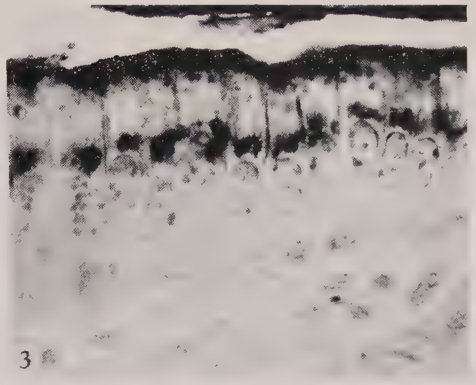
5 Mouse. Small intestine. Formol-saline 12 hours; 10 μ frozen section washed in distilled water 2 hours. Alkaline phosphatase activity present in cuticle, Golgi zone, and a few granules in apical cytoplasm. In the Golgi zone observe the clear ''canalicular'' structures surrounded by region of phosphatase activity.

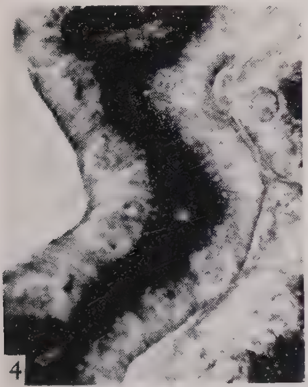
6 Mouse. Small intestine. Formol-saline 3 days, then passed through acetone and benzene into paraffin. Alkaline phosphatase activity is preset in the cuticular border and in the cytoplasm distal to the nucleus, particularly in the supra-nuclear region. The clear spaces immediately distal to the nuclei correspond to the ''canals'' in figure 5.

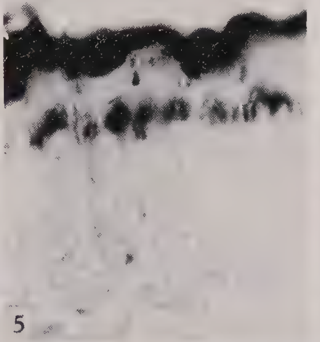
7 Mouse. Kidney. 210 ×. Formol-saline 12 hours, 10 μ frozen section washed in distilled water 4 hours. Alkaline phosphatase is demonstrable in the brush border and lumen of proximal tubules, and in the endothelium of a small artery at the right.

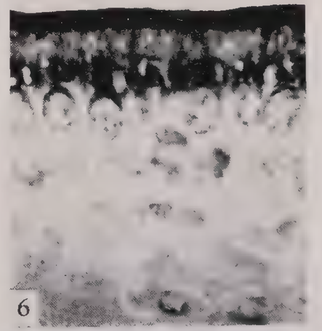
8 Rat. Spleen. 48 ×. Formol-saline 12 hours, 10 μ frozen section washed in distilled water 4 hours. Acid phosphatase activity is prominent in the red pulp and in concentric ''rings'' of macrophages in the white pulp.

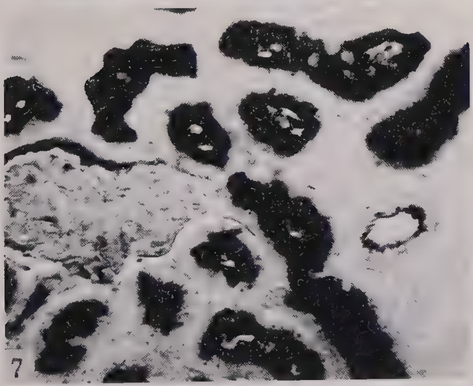

2

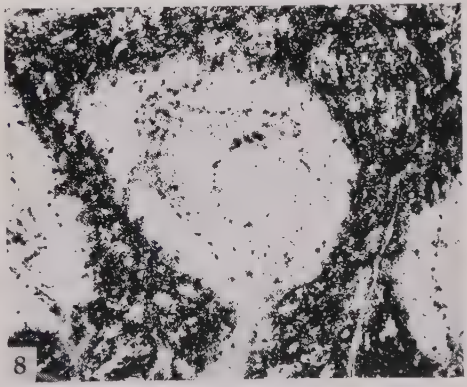

3


4


5


6


7


8

THE AMOEBOID MOVEMENT OF THE MAMMALIAN LEUKOCYTE IN TISSUE CULTURE[1]

P. P. H. DE BRUYN
Department of Anatomy, University of Chicago, Illinois

TWO PLATES (TWELVE FIGURES)

The studies of Mast ('26) and of Pantin ('23) have shown that reversible sol ⇄ gel changes are an essential part of the mechanism of the movement of Amoeba. W. H. Lewis ('31, '39, '42) came to the same conclusion with regard to the movement of leukocytes. Observations on Amoeba under high hydrostatic pressures have provided further evidence for this (Marsland and Brown, '36). Based on these reversible sol ⇄ gel changes, various theories have been proposed to explain the physical force responsible for the protoplasmic movement. Mast believes that the contraction of the plasmagel, which is under elastic tension because of the turgidity of the cell, is responsible for the movement. Pantin holds that swelling resulting from the local production of acid at the anterior end and contraction due to syneresis at the posterior end of the Amoeba cause the protoplasm to be driven forward. Marsland and Brown suggested that volume changes occurring during sol ⇄ gel transformations may lead to a forward flow of the plasmasol. However, in view of Heyman's ('35, '36) dilatometric investigations on sol ⇄ gel changes, this hypothesis was later withdrawn (Marsland, '42). W. H. Lewis ascribes the motion of leukocytes to the "contractile tension, which protoplasm (a colloid) automatically exerts when it gels," (Lewis, '42). This

---

[1] This work was aided by a grant from the Dr. Wallace C. and Clara A. Abbott Memorial Fund of the University of Chicago.

view is based on Lewis' observations on the occurrence of "constriction rings" in moving leukocytes. As the leukocyte moves forward these rings remain stationary relative to points outside the cell. According to this investigator, there is here a local contraction, caused by an increase in the thickness of the superficial gel layer. This layer extends over the body of the cell posterior to the ring. When it contracts the endoplasm and nucleus are forced forward through the constriction ring, which remains stationary.

Observations on moving leukocytes will be reported in this paper which support Lewis' general conclusion that the movement of amoebae and leukocytes is essentially similar in nature. However, the data presented here are difficult to reconcile with that part of his concept which deals with the mechanism of amoeboid movement of leukocytes. For this reason certain modifications in the theory will be proposed so as to bring it in agreement with the observed facts.

MATERIALS AND METHOD

The movement of leukocytes was studied in tissue cultures of buffy coat, lymph node, and bone marrow of rabbits by means of time-lapse photomicrography. The medium consisted of homogenous heparinized plasma, which was stored in sealed ampules, after being frozen and dried. In making the cultures, the dried plasma was redissolved to its original volume with sterile distilled water. No tissue extract was added, since the redissolved frozen-dried plasma clotted rapidly on contact with the explant. The cultures were incubated in the "sitting drop" position but were photographed as hanging drops. Time-lapse photographs were taken with a 4.3-mm fluorite oil immersion objective, Bausch and Lomb, and an 8 × periplan ocular Leitz, on 16-mm reversible film at the rate of one frame per second. When projected at normal rate the motion is speeded up sixteen times, which is too fast for a detailed analysis of leukocyte movement. This analysis was accomplished by projecting the film on paper. Tracings were made from the outlines of the cell of every frame; i.e., at intervals of 1 second.

OBSERVATIONS

The character of the movement of leukocytes in tissue cultures depends on the substrate and also on the cell type (De Bruyn, '44, '45). On the flat surface of the coverglass lymphocytes and heterophil leukocytes move in the general shape of a "handmirror": they have a rounded cell body with an anterior pseudopodial area and a passive tail. They differ in degree of directional polarity. The lymphocytes which move intermittently in locomotory and non-locomotory phases are strictly polarized in the former phase with regard to direction and during this phase they maintain the outline of a handmirror as described above. On the other hand the heterophil leukocytes do not possess so high a degree of polarization and frequently change direction. Their handmirror shape is neither as pronounced nor as constant as that of the lymphocytes: the tail is often withdrawn and, during the changes in direction, the pseudopodial area has a lateral position. For reasons to be discussed later, this study is based on the motion of heterophils during the short periods in which they move in a relatively straight line. The observations on lymphocytes were made during the locomotion phase.

The present work confirms the observations of W. H. Lewis ('31) that the cells moving on a flat surface are attached to the substrate at the anterior pseudopodial area, while the tail and at least part of the cell body are free. In contrast to the highly mobile anterior pseudopodial area, the tail which has a wrinkled surface is relatively rigid. This is clearly seen as the cell changes its direction. The tail, which can be of considerable length, then swings passively around in the opposite direction, without showing any tendency to bend. Inside the plasma clot, the lymphocytes as well as heterophils have a more cylindrical shape and move in a twisting, writhing manner, which has been termed "wormlike" motion.

Constriction rings occur in both types of movement, but they occur with much greater regularity in the cells moving with a wormlike type of motion than in the cells moving in a handmirror shape. Among the latter, there are many cells which do not have or have only occasionally a constriction ring. The wormlike moving cells always have constriction rings. Usually two or three are present at the same time. By projecting the film on paper and carefully marking the places where the cells showed constriction rings, it was found that when two or more cells follow precisely the same path in a culture, they all have constriction rings at precisely the same places in the culture. This clearly indicates that inside the plasma clot the constriction rings are caused by specific configurations of the medium that are external to the moving cell. An example of the evidence supporting this appears in figures 1–4.

According to W. H. Lewis, the cells migrating in the plasma clot sometimes squeeze through the meshes of the fibrin, but he states that the constriction rings are not produced by an external factor, because the same phenomenon occurs when the cells move on the flat surface of the coverglass. In studying the constriction rings of leukocytes moving on the surface of the coverglass, the same procedure was followed as for those moving inside the plasma clot. The place where a constriction ring occurred was again marked and the behavior of leukocytes subsequently passing such a place was observed. It was found for this case also that constriction rings were present at fixed places in the culture (figs. 5–7). Thus there are also obstacles on the coverglass which cause grooves on the surface of the cell. Such obstacles causing a constriction ring are located at such a distance from each other that each side of the cell is in contact with an obstacle.

Not all cells moving along the same path show complete constriction rings; some have only a one-sided indentation. This occurs when the cell is so small that it touches only one of the two points as it passes between them or on the outside of one of them (figs. 9–11). Single obstacles also occur. Then the cells moving along such places, also have only one-sided constrictions. The exact physical nature of these invisible obstacles was not ascertained. They may be formed by the attachment of fibrin fibrils to the glass or possibly by irregularities on the surface of the glass. That they form a considerable resistance to the moving cells can be seen when a cell moves "head-on" towards the obstacle. Then a very deep indentation may occur in the anterior pseudopodial area, almost cutting the cell in two (fig. 8).

When projected at a speed of sixteen frames per second (the motion of the cells is speeded up sixteen times), the sides of the cells moving on the surface seem to exhibit an undulating motion. This gives the impression of fine waves passing over the surface of the cell from the anterior towards the posterior part. This occurs with great regularity in all moving blood cells but is particularly conspicuous in moving lymphocytes. An analysis of this phenomenon by means of tracings made at intervals of one second reveals that actually the "waves" are stationary with respect to points outside the cell (fig. 12). As the cell moves, a lateral part of the protoplasm which forms the anterior pseudopodial area becomes immobilized. While the rest of the cell progresses, this particular part forms a protuberance on the lateral outline of the body of the cell and the tail gradually approaches this protuberance. As this happens, the protuberance diminishes in size and its surface becomes wrinkled. Finally it shrinks away completely and it is taken up in the posterior part of the cell, where the tail joins the cell body. This phenomenon may then occur at the other side of the cell or again at the same side. Also, two protuberances may be present at one time. Most frequently they occur alternately on either side. These lateral protuberances are of varying size. In lymphocytes they may be sometimes so small that they are difficult to analyze by tracings. In the heterophil leukocytes, on the other hand, these lateral protuberances are usually larger than those of the lymphocytes.

The behavior of the granules of heterophils in locomotion is an indication of the absence or presence (and direction) of flow of the cytoplasm. These granules are extremely concentrated in the cell. In addition, they are so minute that it is necessary to use a high magnification objective which results in a loss of depth of focus. These two factors make it difficult to observe one individual granule for a prolonged period. Therefore these observations cannot be made with the same perfection that Mast ('26) was able to obtain in his studies on Amoeba.

Changes of direction of the cell have a marked influence on the direction of flow of the cytoplasm. In heterophil leukocytes such changes of direction or even complete reversals occur very frequently; often within distances shorter than the length of the cell. Because of this it is difficult to establish a correlation between direction of flow and movement. To avoid this complicating factor, these observations are limited to those short periods during which the cell moves in a straight line. Usually the lateral protuberances do not contain any granules and consist only of hyaline cytoplasm. Sometimes, however, a few granules are present. It can then be seen that the granules located in a lateral protuberance remain stationary with regard to objects outside the cell. After the protuberance has contracted, the granules again participate in the streaming, which is in a generally forward direction. Sometimes granules located along the non-protuberant lateral outline of the cell also remain stationary. Immediately medial to these stationary granules, often at a distance of only one granule from the lateral outline, the granules are streaming in a forward direction. The granules which are stationary may remain so until they are reached by the advancing posterior end of the cell; however they may also begin to participate in the streaming before this happens. It is significant that the behavior of these granules in moving heterophil leukocytes shows a striking resemblance to that of the granules and crystals in actively progressing amoebae (Mast, '26).

These phenomena could not be demonstrated in the cells moving inside the culture. Here the outlines of the cells are greatly distorted by the fibrin fibrils of the plasma clot. Observations on these cells are also considerably impaired by the fact that the shape and the position of the cells is such that only part of the whole cell is in focus and can be observed.

DISCUSSION

The above observations indicate that the constriction rings are indentations caused by external factors and therefore cannot be an essential part of the mechanism of the locomotive force in leukocytes. However, Lewis' conclusion, that the essential features of the movement of amoebae and leukocytes are the same for both cell types, is in agreement with the present work. The similarity between the handmirror shape of leukocytes in motion and the shape of moving amoebae has been pointed out before (De Bruyn, '44). Furthermore, the behavior of the lateral protuberances as well as the granules in the heterophil leukocytes indicates that in moving leukocytes, as in moving amoebae, the lateral part of the cytoplasm is stationary.

Adopting the terminology of Mast, it can be said that in leukocytes moving in tissue cultures the highly motile anterior pseudopodial area consists of plasmasol, while the rigid posterior part consists of plasmagel. During locomotion the lateral part of the anterior plasmasol gelates and becomes immobilized. As the cell progresses this part of the plasmagel remains stationary with regard to objects outside the cell until it is reached by the tail end. In the cell body, a solation process must take place, since plasmasol is continuously available at the anterior pseudopodial area. This is essentially in agreement with Mast's observations on the sol ⇄ gel changes in moving amoebae. It differs, however, in that in leukocytes the contraction of the plasmagel has only been observed locally, i.e., in the lateral protuberances. In order to account for the driving forward of the plasmasol by a localized contraction of the plasmagel, it is necessary to assume the presence of a rigid superficial gel covering the cell except at the anterior pseudopodial area. Such a cortical plasmagel has been clearly described in Amoeba proteus by Mast, who found that this gelated cytoplasm was at rest in relation to points in space. This is in agreement with the findings reported in this paper in the sense that the lateral part of the cytoplasm of the leukocytes also remains stationary and hence it is reasonable to assume the presence of a superficial plasmagel layer in leukocytes. W. H. Lewis' hypothesis that leukocytes possess a superficial gel layer, such as Mast has described in Amoeba, is strengthened by the above described observations. The superficial plasmagel may and probably does participate in the process of contraction which occurs in the lateral protuberances, but this is not necessary. The cell is attached to the substrate at the anterior end. The region of attachment includes the newly forming plasmagel. At the posterior edge of this area, the cell becomes free from the substrate. This indicates that the region of the newly formed plasmagel is more adherent to the substrate than that previously formed. Therefore contraction of the superficial plasmagel, either as a whole or locally, will drive the plasmasol and the cell forward.

Lewis ascribed the cause of the contraction to the gelation process itself. It is known that a decrease in volume occurs during the sol → gel transformation of gelatin and agar (Heyman, '35, '36). However the work of Marsland and Brown ('36) indicates that the protoplasmic sols probably do not react in this way when they gelate (see Marsland, '42). The above observations on the lateral protuberances indicate that a contraction does not take place during the sol → gel change, but that the plasmagel itself contracts.

The process of gelation — contraction — solation — gelation, etc., may be compared with the isothermal, reversible sol syneresis described by W. Heller ('42) in thixotropic iron oxide sols. The fluid phase which is separated from the plasmagel during the contraction may then be taken up by the earlier formed plasmagel which changes into plasmasol. Whether the physical basis of this process is actually the same in protoplasm as in the iron oxide sol cannot be decided.

It is of interest to note that the newer concepts of the structure of protoplasm provide an adequate basis for the contraction of the protoplasmic gel as well as for the other changes observed in protoplasm during amoeboid movement. According to most modern workers the molecular structure of protoplasm is constituted by a random arrangement of polypeptide chains which are linked by cross-linkages of the side chains. Water, salts, and lipids are present in the meshes of this molecular network. According to Frey Wysslings' concept ('38), not all side chains are thus bound, but neither are all the side chains loosened and never is the cytoplasm in a true sol state, since some bonds always remain. The side chain system is in constant change: the more bonds that are loosened, the more the cytoplasm approaches the fluid state. In streaming, the side chains would be constantly broken and reunited. In such a system, contraction can be visualized in two ways. First, the locking of more side chains may cause a narrowing of the meshes of this three dimensional reticulum. This will result in contraction. In the second place, a contraction by folding of the polypeptide chains themselves, as has been observed by Astbury ('39), is another possibility.

Both mechanisms will be associated with displacement of water. This water may be taken up by previously contracted cytoplasm through the reverse processes: i.e., breaking up of side-chain linkages and/or springlike extension of folded polypeptide chains. Both will result in a driving forward of the cytoplasm. Such a process is essentially in agreement with Pantin's hypothesis, according to which pseudopod formation is caused by imbibition of water and contraction by syneresis of the plasmagel. The evidence for a differential imbibition of water in amoeboid movement is derived by Pantin from observations on the effect of hypertonic solutions on amoebae. Owing to the abstraction of water in hypertonic solutions the whole amoeba assumes a "radiosa" (type A) or "rugose" (type B) form. Such an appearance under normal conditions is characteristic for the tail-piece. Pantin concludes from this that during amoeboid activity, water is abstracted from the tail-piece, which is imbibed by the cytoplasm at the anterior end and thus causes pseudopod formation. A similar wrinkling of the surface of amoebae due to loss of water has been observed by Mast and Doyle ('33). In moving leukocytes, the parts of the cell which show a wrinkled surface are the tail and the lateral protuberances as these are approached by the posterior end. If this wrinkling of the surface is similarly caused by a loss of water, then it would follow that the contraction of the cytoplasm during amoeboid movement of leukocytes is accompanied by abstraction of water. However, it must be stressed that the gel ⇄ sol transformation, which may occur by reason of behavior of the polypeptide chain with its side chains, is the primary factor in the amoeboid activity of the cell and that the changes in water content of the different parts of the cytoplasm are secondary to this.

SUMMARY

1. The same plasmasol ⇄ plasmagel changes occur in the movement of the mammalian leukocyte as are described by Mast in Amoeba.

2. The "constriction rings" in moving leukocytes are caused by external factors in the culture medium and do not play an essential rôle in the movement of these cells.

3. Observations on moving leukocytes indicate that during locomotion the plasmagel contracts.

4. The possible mechanisms involved in the plasmasol ⇄ plasmagel changes and in the contraction of the plasmagel are discussed briefly.

LITERATURE CITED

ASTBURY, W. T. 1939 X-ray studies of the structure of compounds of biological interest. Ann. Rev. Biochem., vol. 8, p. 113.

DE BRUYN, P. P. H. 1944 Locomotion of blood cells in tissue cultures. Anat. Rec., vol. 89, p. 43.

——— 1945 The motion of the migrating cells in tissue cultures of lymph nodes. Anat. Rec., vol. 93, p. 295.

Frey-Wyssling, A. 1938 Submikroskopische Morphologie des Protoplasmas und seiner Derivate. Borntraeger, Berlin.

Heller, W. 1942 Reversible aggregations of colloidal particles. III. Isothermal reversible sol syneresis. J. Phys. Chem., vol. 46, p. 783.

Heymann, E. 1935 Studies on sol-gel transformations. I. The inverse sol-gel transformation of methylcellulose in water. Trans. Farad. Soc., vol. 31, p. 846.

——— 1936 Studies on sol-gel transformations. II. Dilatometric investigations on iron hydroxide, gelatin, methylcellulose, silicic acid and viscose. Trans. Farad. Soc., vol. 32, p. 462.

Lewis, W. H. 1931 Locomotion of lymphocytes. Bull. Johns Hopkins Hosp., vol. 49, p. 29.

——— 1939 The role of a superficial plasmagel layer in changes of form, locomotion and division of cells in tissue cultures. Arch. f. exper. Zellforsch, vol. 23, p. 1.

——— 1942 The relation of the viscosity changes of protoplasm to ameboid locomotion and cell division. The Structure of Protoplasm. Am. Soc. Plant. Physiol., Iowa State College Press, Ames, Iowa.

Marsland, D. A. 1942 Protoplasmic streaming in relation to gel structure in the cytoplasm. The Structure of Protoplasm. Am. Soc. Plant Physiol., Iowa State College Press, Ames, Iowa.

Marsland, D. A., and D. E. S. Brown 1936 Amoeboid movement at high hydrostatic pressure. J. Cell. and Comp. Physiol., vol. 8, p. 167.

Mast, S. O. 1926 Structure, movement, locomotion and stimulation in Amoeba. J. Morph., vol. 41, p. 347.

Mast, S. O., and W. L. Doyle 1934 Ingestion of fluid by Amoeba. Protoplasma, vol. 20, p. 555.

Pantin, C. F. A. 1923 On the physiology of amoeboid movement. I. J. Marine Biol. Assoc., vol. 13, p. 24.

EXPLANATION OF PLATES

The halftones are enlargements from time-lapse photographs on 16-mm film with 4.3 mm fluorite oil immersion objective, Bausch and Lomb and 8 × periplan ocular, Leitz. Magnification: 1200 ×.

The line drawing is obtained by projecting the film on paper and tracing the outlines of the cell of every frame, i.e., at intervals of 1 second. Magnification: 840 ×.

None of the cultures was older than 8 hours.

PLATE 1

EXPLANATION OF FIGURES

1–4 Culture of rabbit bone marrow. Two heterophil leukocytes moving inside the plasma clot.

1, 2 Heterophil leukocyte advancing from left to right through a ''constriction ring.''

3, 4 Another heterophil leukocyte advancing from left to right, showing a ''constriction ring'' at exactly the same place in the culture as the cell in figures 1 and 2.

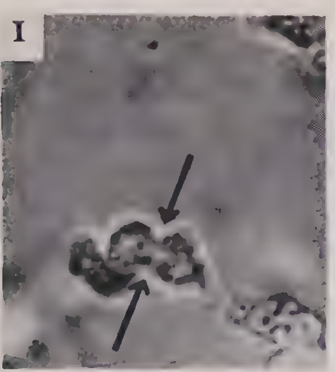
5–8 Culture of rabbit buffy coat. Four lymphocytes moving on the surface of the coverglass.

5 Lymphocyte advancing from right to left through a ''constriction ring.''

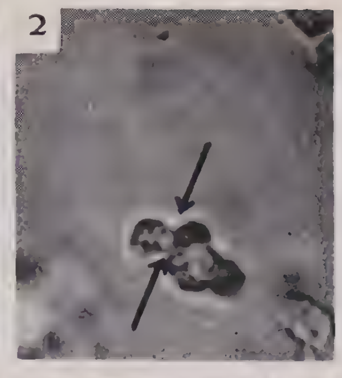
6 Another lymphocyte advancing from right to left, showing a ''constriction ring'' at exactly the same place in the culture as the cell in figure 5.

7 A third lymphocyte advancing from right to left, showing a ''constriction ring'' at exactly the same place in the culture as the cells in figures 5 and 6.

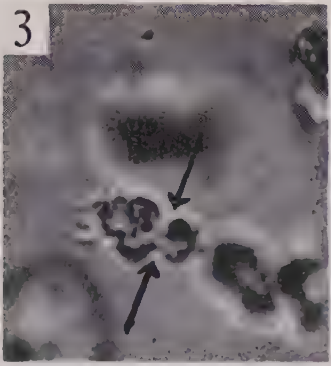
8 Hypertrophied lymphocyte moving from the left ''head-on'' towards the obstacle, which produced the lower indentation of the ''constriction ring'' in each of the lymphocytes in figures 5, 6 and 7.



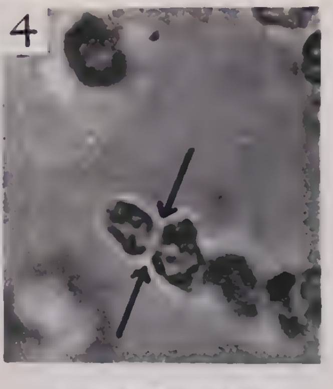
1



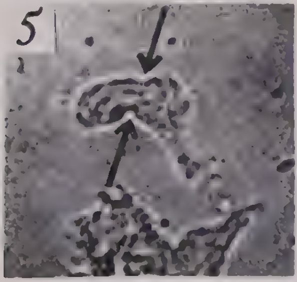
2



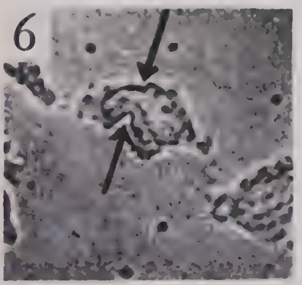
3



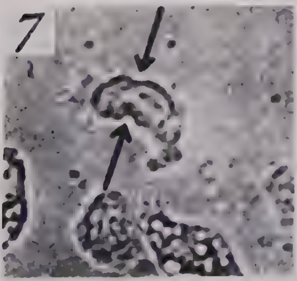
4



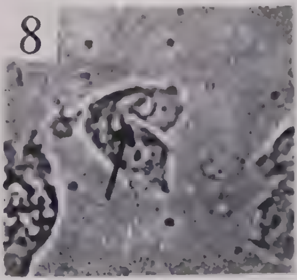
5



6



7



8

PLATE 2

EXPLANATION OF FIGURES

9–11 Culture of rabbit buffy coat. Two heterophil leukocytes (figs. 9 and 11) and one lymphocyte (fig. 10) moving on the surface of the coverglass.

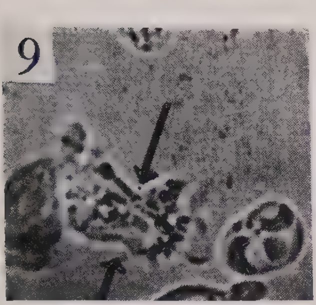
9 Heterophil leukocyte moving from left to right through a ''constriction ring.''

10 Lymphocyte moving from above to below, having a one-sided indentation at exactly the same place in the culture where the upper indentation of the ''constriction ring'' of the heterophil leukocyte in figure 9 occurred.

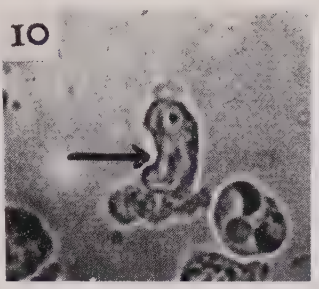
11 Heterophil leukocyte moving from below to above, having a one-sided indentation at exactly the same place in the culture where the indentation of the lymphocyte in figure 10 and the upper indentation of the ''constriction ring'' of the heterophil leukocyte in figure 9 occurred.

12 Tracing of a lymphocyte in a culture of rabbit lymph node. The cell moves from S (S′, S″) to the right. a–a, b–b, c–c, d–d: stationary lateral protuberances (see text). S, S′, S″ are continuations of the same line.

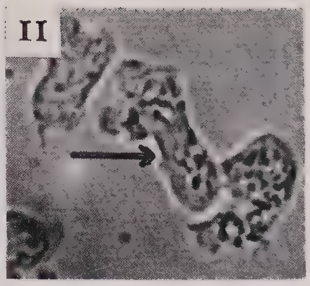
9



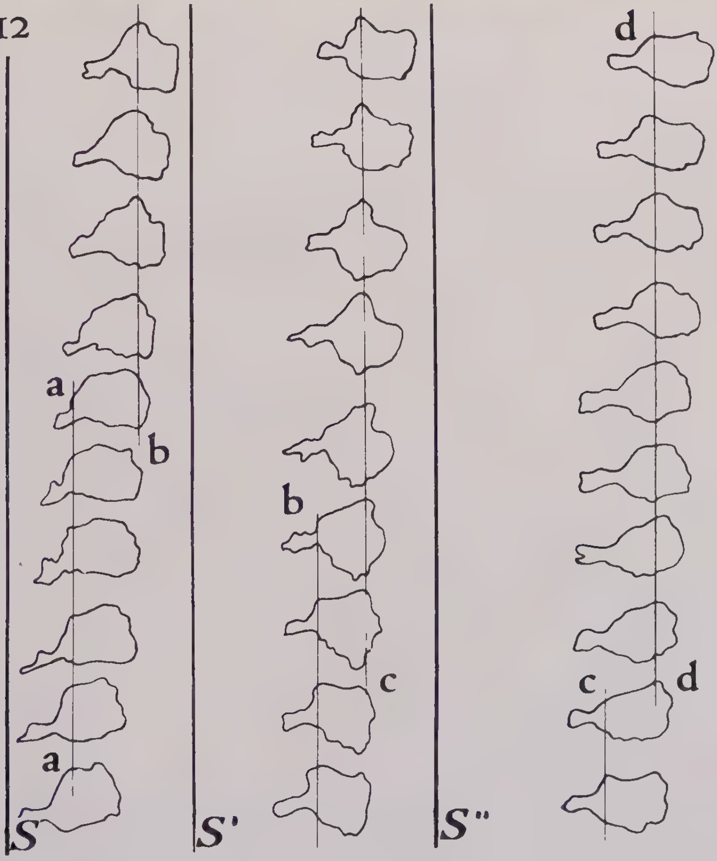
10



11



12


a
b
a
b
c
d
c
d
S
S'
S"

GLYCOGEN AND PHOSPHATASE IN THE DEVELOPING HAIR

P. L. JOHNSON AND G. BEVELANDER
Departments of Anatomy, College of Dentistry and The Graduate School of Arts and Science, New York University

ONE PLATE (EIGHT FIGURES)

INTRODUCTION

Investigations pertaining to the relationship existing between the localization of the enzyme alkaline phosphatase and glycogen have been reported in connection with developing bone by Harris ('32), Glock ('40), and Horowitz ('42). Similar observations detailing these relationships in the placenta and endometrium have been reported by Dempsey and Wislocki ('44, '45), and by Wislocki and Dempsey ('45). Bevelander and Johnson have described the histochemical localization of alkaline phosphatase ('45), and glycogen ('46), in connection with the development of teeth. Johnson et al. ('45), have further investigated the distribution of phosphatase during the cyclic growth of the hair of the rat.

This present investigation is a continuation of our previous studies and was undertaken in order to establish a correlation between the phosphatase and glycogen interrelationships during the development of hair, and secondly to examine the possible role which these two substances play during the various stages of growth and differentiation.

MATERIAL AND METHODS

These studies are based on pig embryos obtained from freshly slaughtered pigs. The embryos ranged in size from 20 mm to 240 mm. The developmental stages of the hair were selected from various regions of the face and jaw.

Phosphatase was determined by the method of Gomori ('41a) and glycogen by the Gage Iodine method and checked by the silver method of Mitchell and Wislocki ('44).

In addition to the hair of the pig which we have investigated in detail, sections of the hair of the rat, and rabbit in various stages of growth were also examined for the localization of glycogen.

OBSERVATIONS

Before the hair bud differentiates, both glycogen and phosphatase are found simultaneously in the primitive epidermis. As soon as the hair bud appears however, phosphatase is found to be somewhat more abundant in the basal layer of cells than in the surface layers. Phosphatase is however most abundant in the underlying condensation of mesenchyme which represents the presumptive papilla, figure 1. The glycogen distribution at this stage of development shown in figure 2, is confined exclusively to the epidermis.

In figure 3, a slightly more advanced stage in the development of the bud, the diminution of phosphatase in the epithelial component of the primitive hair is very marked when compared with the slightly earlier stage shown in figure 1. The glycogen distribution in a bud having undergone approximately the same amount of differentiation, shows that the glycogen is still confined exclusively to the proliferating epithelial bud, figure 4. In the subsequent growth of the hair, the epithelial bud undergoes considerable elongation until eventually the primitive hair follicle consists of an inner and outer epithelial layer, both lying within a connective tissue sheath. At the base of the hair follicle meanwhile, the mesenchymal tissue has invaded the bulb to give rise to th papilla. In a later stage of development, rapid mitotic activity of the epithelial cells in the region known as the matrix results in the production of the hair shaft, located internal to the epithelial layers.

In this first phase of hair growth just described, we observe the following: Phosphatase is very prominent in the papilla, in the external connective tissue sheath, and to a lesser degree in the nuclei of the cells of the matrix where mitotic activity is taking place, figure 5. A comparable stage in development treated to show glycogen distribution, figure 6, reveals that this substance is abundantly present in the outer and inner epithelial sheaths of the hair, present to some extent in the external connective tissue sheath, but completely lacking in the papilla and matrix.

As the hair erupts, we observe a localization of phosphatase and glycogen represented in figures 7 and 8 respectively. In the specimen shown in figure 7, a section of a growing erupted hair, the same relationships in regard to phosphatase distribution was observed as occurs in the somewhat earlier stage of development, that is, it occurs in abundance in the external connective tissue sheath and papilla, while in the cells of the matrix it is present to only a slight degree. The glycogen distribution on the other hand, as shown in figure 8, is confined exclusively to the internal and external epithelial layers.

DISCUSSION

Phosphatases are now known to be present in a wide variety of both embryonic (Moog, '44) and adult tissues (Gomori, '41a, '41b; Kabat and Furth, '41; Bourne, '43). These enzymes are moreover known to be concerned with carbohydrate, phospholipid and nucleotide metabolism as well as with the process of calcification (Sumner and Somers, '43).

The chemical events which lead to the formation of glycogen are now believed to consist in a series of steps in which phosphate is split from some carbohydrate moiety. In this connection (Wislocki and Dempsey, '45). have formulated an interesting hypothesis. They suggest that due to the wide spread occurrence of phosphatase it seems likely that in many tissues this enzyme is utilized in the process of dephosphorylation. They further suppose that if phosphatase is utilized in this manner, then phosphatase activity represents the ''penultimate'' stage in glycogenesis. This fact they hold may be experimentally demonstrated if it can be shown that phosphatase intervenes between the source of the hexose phosphate and the region in which glycogen is being deposited.

The observations of these authors in regard to the placenta and the uterus lead them to conclude "that a region containing phosphatase is invariably interposed between the blood stream and the locality in which glycogen is deposited." They further point out, that the above relationship is not constant and list several instances in which glycogen and phosphatase are both present within the same cell.

In the phosphatase-glycogen relationships of the developing hair we have noted the following: First of all, both phosphatase and glycogen are present in abundance in the epidermis. As the epithelial bud elongates and differentiates, glycogen comes to be localized chiefly in the epithelial components, while phosphatase is localized in the mesenchymal derivatives. If we now consider the same features in the later developmental stages of the hair, one finds that by the time the hair erupts, phosphatase occurs adjacent and external to the deposited glycogen.

Our observations on developing hair reveals that the spacial relationship between phosphatase and glycogen correlates well with the path of glucose entry into the external and internal epithelial sheaths of the hair. In the developing tooth however, we have observed (Bevelander and Johnson, '45, '46), that while glycogen is confined to the epithelial component of the enamel organ, phosphatase is likewise present in this same epithelial tissue, a situation comparable to that observed on occasion by Wislocki and Dempsey.

SUMMARY

1. Both alkaline phosphatase and glycogen are present in the primitive hair bud.

2. On elongation, phosphatase disappears from the bud while glycogen remains abundant. Phosphatase makes its appearance in the mesenchymal components of the hair as soon as they are elaborated.

3. During the later stages of differentiation and growth leading to the eventual eruption of the hair, the spatial relations between phosphatase and glycogen correlate well with the path of glucose entry into the tissues.

LITERATURE CITED

BEVELANDER, G., AND P. L. JOHNSON 1945 The histochemical localization of alkaline phosphatase in the developing tooth. J. Cell. and Comp. Physiol., vol. 26, pp. 25–33.

——— 1946 The histochemical localization of glycogen in the developing tooth (In press).

BOURNE, G. 1943 The distribution of alkaline phosphatase in various tissues. Quart. J. Exp. Physiol., vol. 32, pp. 1–20.

DEMPSEY, E. W., AND G. B. WISLOCKI 1944 Observations on some histochemical reactions in the human placenta, with special reference to the significance of the lipoids, glycogen and iron. Endocrinology, vol. 35, pp. 409–429.

——— 1945 Histochemical reactions associated with basophilia and acidophilia in the placenta and pituitary gland. Am. J. Anat., vol. 76, pp. 277–301.

GLOCK, G. E. 1940 Glycogen and calcification. J. Physiol., vol. 98, pp. 1–11.

GOMORI, G. 1941a The distribution of phosphatase in normal organs and tissues. J. Cell. and Comp. Physiol., vol. 17, pp. 71–83.

——— 1941b Distribution of acid phosphatases in the tissues under normal and pathologic conditions Arch. Path., vol. 32, pp. 189–199.

HARRIS, H. A. 1932 Glycogen in cartilage. Nature, vol. 130, pp. 996–997.

HOROWITZ, N. H. 1942 Histochemical study of phosphatase and glycogen in fetal heads. J. Dent. Res., vol. 21, pp. 519–527.

JOHNSON, P. L., E. O. BUTCHER AND G. BEVELANDER 1945 The distribution of alkaline phosphatase in the cyclic growth of the rat hair follicle. Anat. Rec., vol. 93, pp. 355–362.

KABAT, E. A., AND J. FURTH 1941 A histochemical study of the distribution of alkaline phosphatase in various normal and neoplastic tissues. Am. J. Path., vol. 17, pp. 303–318.

MITCHELL, A. J., AND G. B. WISLOCKI 1944 Selective staining of glycogen by ammoniacal silver nitrate. Anat. Rec., vol. 90, pp. 261–266.

MOOG, F. 1944 Localizations of alkaline and acid phosphatases in the early embryogenesis of the chick. Biol. Bull., vol. 86, pp. 51–80.

SUMNER, J. B., AND G. F. SOMERS 1943 Chemistry and Methods of Enzymes. Academic Press Inc., New York.

PLATE 1

EXPLANATION OF FIGURES

The photographs shown in plate 1 are reproductions of sections of developing hair follicles in the skin of the jaws of foetal pig embryos. The darkened areas in figures 1, 3, 5, and 7 represent the localization of alkaline phosphatase. The darkened areas in figures 2, 4, 6, and 8 represent the localization of glycogen.

1 Section of a primitive hair follicle showing distribution of phosphatase in the epidermis and the presumptive papilla.

2 Primitive follicle comparable to the one shown in figure 1, treated to show the distribution of glycogen.

3 Hair follicle slightly more advanced than the one shown in figure 1. Note the loss of phosphatase in the epithelium.

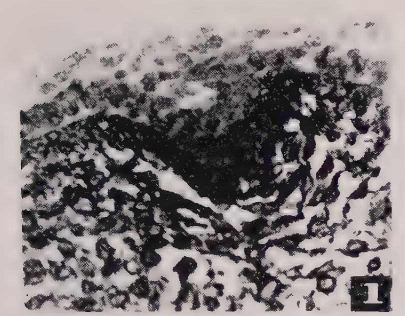
4 Section of follicle corresponding in development to the one shown in figure 3. Glycogen confined to epithelium only.

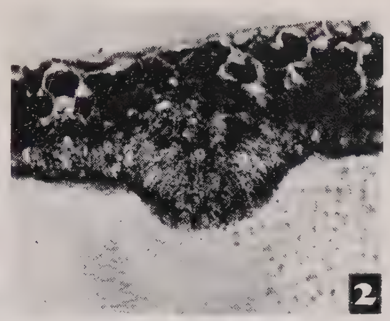
5 Hair follicle much elongated showing phosphatase confined to the papilla, external sheath, and to cells adjacent to the papilla.

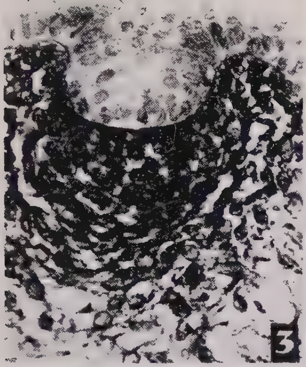
6 Section similar to that shown in figure 5, to show glycogen distribution.

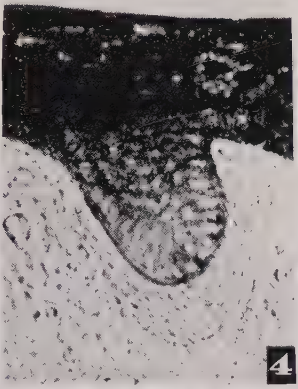
7 Erupted hair treated to show phosphatase distribution. The dark stain shown in hair shaft is due to pigment.

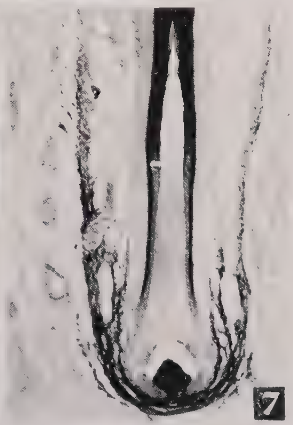
8 Erupted hair treated to show glycogen distribution. At this stage of development the glycogen is confined to the external and internal epithelial sheaths.

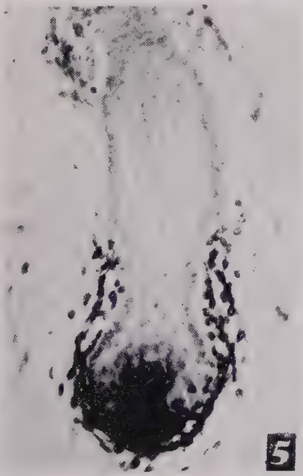

1

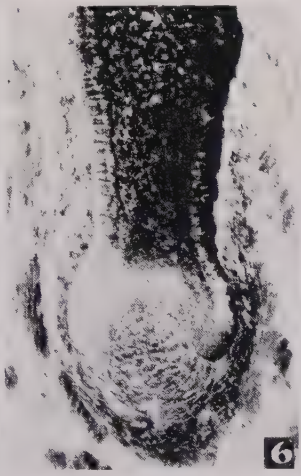

2

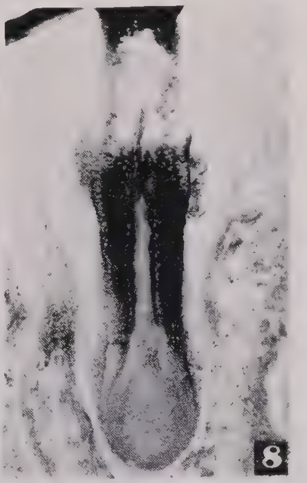

3


4


7


5


6


8

PROCEEDINGS OF THE AMERICAN ASSOCIATION OF ANATOMISTS

FIFTY-NINTH SESSION

Western Reserve University, School of Medicine, Cleveland, Ohio
April 4, 5 and 6, 1946

After 3 years, during which annual meetings were cancelled because of transportation difficulties caused by World War II, the Association held its Fifty-ninth Meeting in Cleveland, Ohio, by invitation of Western Reserve University School of Medicine, Thursday, April 4, Friday, April 5, and Saturday, April 6. The demonstrations were held at the Medical School; all other sessions at the Hotel Statler. The Local Committee was composed of Samuel W. Chase, William C. Barrett, Ernst Scharrer, C. C. Adams, Arnold Lazarow, Holly Broadbent and Normand Hoerr, Chairman.

Registered Attendance:

	1946	1942		1946	1942
Association members	308	226	Out of town	386	309
Non-members	104	201	Local	26	118
	412	427		412	427

RESUMÉ OF THE SCIENTIFIC PROGRAM

The Fifty-ninth session of the American Association of Anatomists was opened in general session by Acting President J. Parsons Schaeffer at 10:00 A.M. in the Euclid Ballroom of the Hotel Statler. Following the announcements of

Auditing and Nominating Committees, seven papers were presented of which five were scientific papers read from platform, the sixth a motion picture, and the seventh a talk on "Anatomists and international affairs" by one of our members who is also a member of the Department of State at Washington, D. C.

At 12:00 NOON, the *Presidential Address* was delivered by Professor J. Parsons Schaeffer on the subject:

"Anatomy today in the medical curriculum."

The remainder of the program was divided into (a) Sessions for the reading of papers: Thursday afternoon, 2:00 to 5:00; Friday morning, 9:30 to 12:15; Saturday morning, 9:30 to 1:00; (b) a session of demonstrations Friday afternoon, 2:00 to 5:30; (c) a session for motion picture demonstrations Thursday evening, 7:00 to 9:45; and (d) a general session with an invitation program Saturday afternoon, 2:00 to 4:00. On each of the three sessions, Thursday afternoon, Friday and Saturday mornings, there were four concurrent programs scheduled. (Regarding days of the week, an attempt was made to schedule the meetings Wednesday, Thursday and Friday, but it was impossible to obtain the hotel for Wednesday. The hotel situation also determined the shift of the meeting away from the week preceding Easter, which occurred on April 21, 1946.)

Again, as in 1942 at the New York meeting, the demonstrations were restricted to a single afternoon because of the distance of the Western Reserve University Laboratories from the hotel where the papers were presented. This was unfortunate, for it resulted in congestion in some of the demonstration rooms and allowed too little time for this important part of the program. However, the placing of the motion pictures in the evening left the general demonstrations without competition.

Attractive and instructive features of the demonstrations were the displays of the museums and of work in progress in the very live Department of Anatomy at Western Reserve. In addition to the Hamann Museums of Anatomy and Comparative Anthropology, some of the products of the Brush Foundation for the purpose of studies leading to the betterment of the human stock, and apparatus, charts and photographs which have developed under the Bolton Fund for the study of the growth of face and jaws, much of which was inaugurated by the indefatigable T. Wingate Todd, there was demonstrated extensive equipment for studies in the field of cytochemistry, which is of especial interest to Professor Hoerr.

The Anatomists were invited to visit the Cleveland Medical Library where rare books of the Army Medical Library have been housed during the war. Some of the choice anatomical items were displayed in one of the demonstration rooms.

The scheduling of motion pictures in the evening preceding the Smoker worked out satisfactorily except that the eight reels made the program rather long — 7:15 to about 9:45; in spite of the transfer of the longest reel to the Thursday morning session.

An attempt was made to have the pictures sent to the Local Committee for previewing. This attempt was only partially successful, since two or three films were permitted, for various reasons, to be shown without previewing. In the light of the present excessive activity of anti-vivisectionists, it is believed that great caution should be exercised in this matter unless attendance at the motion pictures is restricted to members only.

Number of Papers and Demonstrations

	1946	1942	1941
Papers from platform	144	134	132
Papers withdrawn	3	3	8
Papers actually presented	141	131	124
Papers read by title	46	51	32
Demonstrations (regular)	51	46	50
Motion pictures as demonstrations	8	5	7
Motion pictures as papers	1	2	2
Motion pictures by title	0	2	2

For the Friday afternoon invitation program, Acting President Schaeffer arranged the following:

E. V. Cowdry:[1] Correlated microscopic and chemical changes in epidermal carcinogenesis in mice.

G. L. Streeter: The syndromic nature of organogenesis in the human embryo.

Paul A. Weiss: Frozen-dried tissues as grafts.

SOCIAL ACTIVITIES

Banquet. Attendance (291) exceeded by 46 all previous records. Acting President Schaeffer as toastmaster introduced Dr. Normand L. Hoerr, who as Professor of Anatomy at Western Reserve School of Medicine, welcomed the Association. Dr. Webster G. Simon, vice-present of Western Reserve University, also welcomed the Association, and spoke briefly on the development of research both to satisfy man's innate curiosity and to develop new facts for the betterment of mankind. Dr. Franz Weidenreich, in part as representative of the Society of Physical Anthropologists, which had held its annual meeting in Cleveland on the two days preceding the Anatomists' meeting, gave a prepared address on the relation between physical anthropology and anatomy. The final speaker was president-elect, G. W. Corner, who gave a witty and inspiring talk. "Our group," he said, "must be one in which youth is always welcome and age and experience respected, in order that we may find solutions for old problems and discover truths in the battle against ignorance and disease."

---

[1] From the Barnard Free Skin and Cancer Hospital and the Department of Anatomy, Washington University.

Smoker. Approximately 350 members enjoyed the Smoker, the expenses of which, at the insistence of the Executive Committee, was for the first time borne by the Association.

Tea. A most delightful tea was arranged by the wives of faculty members at the Medical School from 4:00 to 5:00 in the afternoon of the demonstrations.

MINUTES OF THE BUSINESS SESSION

Thursday, April 4, 1946.

10:00 a.m. At a brief Business Session, Acting President Schaeffer announced the appointment of the following committees:

Auditing Committee: Samuel W. Chase, Chairman, A. J. Ramsay.

Nominating Committee for 1947: Bradley M. Patten, Chairman, Davenport Hooker, Eben J. Carey.

Friday, April 5, 1946.

12:25 p.m. Principal Business Session, Acting President Schaeffer in the chair. The President reported that the minutes of the Fifty-eighth Session were printed in The Anatomical Record, vol. 83, pp. 131–137 and 141–155, May 25, 1942, reprints of which had been distributed to all members, and would not be read unless called for. He further reported that interim business of the Association had been reported in annual Proceedings, printed in The Anatomical Record as follows: 1943, vol. 86, pp. 593–645; 1944, vol. 89, pp. 361–374; and 1945, vol. 92, pp. 305–325, reprints of all of which had been distributed to all members, and suggested that they be approved along with the minutes of the 1942 meeting. On motion duly made and seconded, the minutes and the interim reports were approved as printed.

Report of the Treasurer: The Secretary-Treasurer made the following report for the year 1945:

1. (a) *Financial Statement for the year 1945*

Balance on hand in checking account, December 31, 1944 in First National Bank, Philadelphia, Pennsylvania	$2,182.21	
Receipts from dues	875.93	
Total credits		$3,058.14
Expenditures		
Postage, stationery, supplies and miscellaneous	79.67	
Wistar Institute		
Abstracts and postage	411.05	
Proceedings, postage and portrait (S. H. Gage)	194.48	
Printing	43.25	
Contribution to Union of American Biological Societies	10.00	
First National Bank		
Delayed checks	6.25	
Charges for handling account	12.78	
Total expenditures		757.48
		$2,300.66
Assets		
Balance on hand December 31, 1945 and deposited in the name of the American Association of Anatomists in the First National Bank, Philadelphia, Pennsylvania	$2,300.66	
War Bond Defense Series F dated May, 1942 in safe deposit box, First National Bank, Philadelphia, Pennsylvania Value December 31, 1945 $760.00	760.00	
Total assets		$3,060.66

Total assets increased by $118.45 during the year. (Note: In this report as read at the meeting, the value of the war bond on December 31, 1945, was given as $776.00. On reinvestigation the Secretary found that in this and the two previous reports slight errors had been made in the calculated increase in value of the bond. The above report has been corrected accordingly.)

Report of the Auditing Committee: Dr. Chase read the following report:

"The undersigned committee has examined the accounts of the Secretary-Treasurer for the year 1945 and has found total receipts to be those entered in the journal, expenditures in accordance with receipted bills, and the balance on hand December 31, 1945 in agreement with the bank statement of that date."

(Signed)

Samuel W. Chase
Andrew W. Ramsay

On motion made and seconded the reports of the Treasurer and of the Auditing Committee were accepted and adopted.

Election of Officers: The Nominating Committee, consisting of Harold Cummins, Chairman, K. E. Mason and W. F. Windle, placed the following nominations before the Association:

For President, George W. Corner; for First Vice-President, Edward A. Boyden; for Second Vice-President, Marion Hines, each for a two year term. For Secretary-Treasurer, Normand L. Hoerr for a term of four years. For members of the Executive Committee for terms expiring in 1950, William Bloom and William W. Greulich.

In the absence of additional nominations, a motion was made, seconded and carried that the Secretary cast a unanimous ballot for the above-named nominees, and they were declared elected.

Election of Representatives and Committee Members: The Secretary presented, in behalf of the Executive Committee, the following nominations: Representatives to the Council of the American Association for the Advancement of Science for 1 year, J. L. Bremer and F. T. Lewis. Representative to the National Research Council for three years, C. C. Speidel. Representative on the Commission for Standardization of Biological Stains, H. A. Davenport. Delegate to the Union of Biological Societies for 3 years, S. R. Guild. Motion Picture Committee, W. H. Lewis, C. C. Speidel, E. R. Clark, Chairman.

In the absence of other nominations, the above nominees were duly elected.

The Association voted to discontinue with thanks the Committee of National Defense.

Election of New Members: The names of all persons chosen by the Executive Committee in 1943 (17), 1944 (33) and 1945 (29) as "provisional members" were placed in the hands of Association members on mimeographed sheets. Their names had been published as "provisional members" in the Proceedings of the respective years, awaiting action in a regular annual meeting as specified in the Constitution. All 79 were unanimously elected to membership.

The Secretary then presented the following 33 names of persons recommended by the Executive Committee for election to membership in the Association, the names in parentheses indicating the sponsors:

ANDREWS, FREDERICK N., B.S., M.S., Ph.D., Assoc. Prof. Animal Husbandry, Purdue. (F. F. McKenzie, M. D. Overholser.)

ASHLEY, LAURENCE M., B.S., A.M., Ph.D., Instr. Microscopic Anatomy, Med. Evangelists. (E. Gardner, G. H. Scott.)

BACON, ROBERT LEWIS, B.S., M.S., Ph.D., Instr. Anatomy, Stanford. (C. H. Danforth, W. W. Greulich.)

BERGMANN, LOUIS LAWRENCE, M.D., Instr. Anatomy, New York Univ. Coll. of Med. (E. W. Dempsey, D. Sheehan.)

DOYLE, WILLIAM LEWIS, M.A., Ph.D., Assoc. Prof. Anatomy, University of Chicago. (W. Bloom, R. S. Snider.)

EDDS, MAC VINCENT, JR., B.A., M.A., Ph.D., Instr. Anatomy, Univ. of Pittsburgh. (P. Weiss, D. Hooker.)

GLICKMAN, IRVING, B.S., D.M.D., Assist. Prof. Oral Pathology, Tufts Dental School. (G. F. Sykes, E. A. Joy.)

GLUECKSOHN-SCHOENHEIMER, SALOME, Ph.D., Res. Assoc., Columbia University. (D. Rudnick, J. S. Nicholas.)

HEGRE, ERLING S., B.A., M.S., Ph.D., Assoc. Prof. Anatomy, Med. Coll. of Virginia. (H. L. Osterud, A. D. Brashear.)

JOHNSON, PERCY L., B.S., M.S., Ph.D., Asst. Prof. Anatomy, New York University Dental College. (G. Bevelander, E. O. Butcher.)

KAPLAN, EMANUEL B., B.S., M.D., Instr. in Anatomy, College of P. and S., Columbia University. (S. R. Detwiler, P. E. Smith.)

KOLOUCH, FRED, JR., A.B., M.A., M.B., M.D., Fellow, Nat. Sci. Found., Univ. Minnesota. (H. Downey, B. Campbell.)

LAUER, EDWARD W., B.S., M.A., Ph.D., Instr. Anatomy, Michigan. (B. M. Patten, E. C. Crosby.)

LOBO, BRUNO ALIPIO, B.A., M.D., "Docente Livre" Professor Histology and Embryology, University of Brazil. (P. E. Smith, S. R. Detwiler.)

LONG, MARGARET ELEANOR, Ph.D., Instr. Anatomy, Med. Coll. of Alabama. (J. B. Looper, C. M. Goss.)

MACFARLAND, WILLIAM E., B.A., Ph.D., Editor, W. B. Saunders & Co. (R. F. Becker, H. A. Davenport.)

MARBURG, OTTO, M.D., Clinical Prof. Neurology, College of P. & S., Columbia. (F. A. Mettler, A. Elwyn.)

MARVIN, HORACE NEWELL, B.A., M.A., Ph.D., Asst. Prof. Anatomy, Arkansas (R. K. Meyer, B. L. Robinson.)

MAYER, EDMUND, M.D., Pathologist, Chemotherapy Division, Stamford Res. Labs., Amer. Cyanamid Co. (F. T. Lewis, G. B. Wislocki.)

Parshley, Mary Stearns, A.B., M.A., Ph.D., Res. Assoc., Dept. of Pathology, Coll. of P. & S., Columbia. (E. R. Clark, R. G. Williams.)

Phillips, Howard M., B.S., M.A., Ph.D., Professor Biology, Emory University. (J. H. Venable, H. W. Ades.)

Rasmussen, Grant Litster, Ph.D., Assoc. Prof. Anatomy, Buffalo. (A. M. Lassek, A. T. Rasmussen.)

Ring, John Robert, A.B., Sc.M., Ph.D., Instr. in Micro-anatomy, Saint Louis Univ. (A. Kuntz, W. F. Alexander.)

Roosen-Runge, Edward C., M.D., Pathologist, Wrentham State School, Wrentham, Mass. (W. H. Lewis, E. J. Farris.)

Sicher, Harry, M.D., Prof. of Anatomy and Histology, Loyola Univ. School of Dentistry. (I. Schour, O. F. Kampmeier.)

Sosa, Julio Maria, M.D., Head, Div., Hist. Res., Asst. Prof. Hist. and Embryo., Univ. of the Republic, Montevideo, Uruguay. (R. R. Bensley, B. Campbell.)

Spofford, Walter R., B.S., Ph.D., Asst. Prof. Anatomy, Vanderbilt. (S. L. Clark, J. W. Ward.)

Spratt, Nelson T., Jr., A.B., Ph.D., Instr. Biology, Res. Asst. Embry., Johns Hopkins. (B. H. Willier, R. K. Burns.)

Tallmadge, G. Kasten, A.M., M.D., Ph.D., Asst. Prof. Anatomy, Marquette. (E. J. Carey, W. E. Sullivan.)

Taylor, A. Cecil, B.A., M.A., Ph.D., Asst. Prof. Anatomy, Coll. Dentistry, New York University. (E. O. Butcher, G. Bevelander.)

Tunturi, Archie Robert, A.B., M.S., Ph.D., Asst. Prof. Anatomy, University of Oregon. (O. Larsell, W. F. Allen.)

Weber, Neal A., B.A., M.A., M.S., Ph.D., Assoc. Prof. Anatomy, Univ. of North Dakota. (H. E. French, A. B. Dawson.)

Wright, Philip Lincoln, B.S., M.S., Ph.D., Asst. Prof. Zoology, Montana. (R. K. Meyer, H. W. Mossman.)

(Note: The mimeographed sheets giving names of nominees carried 34 names. It was found later that one of the nominees, Charles M. Berry, had been included in the 1944 list, leaving 33 nominees for 1946.)

On motion, duly seconded and passed unanimously, the Secretary was instructed to cast a ballot for all candidates proposed by the Executive Committee, and they were declared elected.

(For information, the Secretary reports that during the past 4 years, there have been 20 additional nominations, 3 of which were withdrawn, 17 acted upon unfavorably by the Executive Committee.)

Members placed in the category of "members relieved of the payment of dues": The Secretary reported that, by action of the Executive Committee, 3 members had been placed in this category in accordance with the provisions of Article VI, Section II of the Constitution, which reads: "Any member who has paid the annual dues for 30 years, or who has attained the age of sixty-five years, or who has retired because of illness, may be relieved of the payment of the annual dues at the discretion of the Executive Committee." It was stated that this brings to 30 the total number of persons in this category.

Members deceased: The Secretary presented the following statement of the Executive Committee:

The Association has suffered very great loss through the death during the year of the following six members:

GEORGE STEPHEN DE RÉNYI, born April 4, 1892, died August 24, 1945. Elected to membership in 1927; Associate Professor of Anatomy, University of Pennsylvania. Made valuable additions to our knowledge of cell structure as revealed by microdissection; an inspiring teacher of histology and embryology.

HENRY SEELYE EMERSON, born March 21, 1907, died March 8, 1946. Elected to membership in 1941. Assistant Professor of Biology, Amherst College. An outstanding investigator and teacher. Made a series of notable contributions in the field of experimental embryology, by the use of an original method: namely, the transplantation of embryonic tissues into regenerating larval tissue.

FRANK BLAIR HANSON, born July 15, 1886, died July 21, 1945. Elected to membership in 1921. Formerly Professor of Zoology, Washington University; recently Associate Director, Rockefeller Foundation. Made important contributions to genetics.

CLARENCE ERWIN MCCLUNG, born April 5, 1870, died January 7, 1946. Elected to membership in 1915. Formerly Director, Zoological Laboratory, University of Pennsylvania; after retirement, during the war, Acting Head, Department of Biology, Swarthmore. One of America's leading zoologists. Contributed epoch-making studies on chromosomes.

BERTRAM GARNER SMITH, born October 7, 1876, died July 30, 1945. Elected to membership in 1921. Professor of Anatomy at New York University until his retirement in 1942. Made valuable contributions in the field of embryology.

James Thomas Wilson, born April 14, 1861, died September 2, 1945. Elected to membership in 1913. Emeritus Professor of Anatomy at University of Cambridge, England; earlier, for 33 years Professor of Anatomy at Sydney, Australia. Conspicuous as a teacher, investigator and administrator. Made valuable studies on the anatomy of marsupials and monotremes, and in the field of embryology.

Members Resigned:

Raymond A. Dart, Professor of Anatomy and Dean of Faculty of Medicine, University of Witwatersrand, South Africa.

Paul Huston Stevenson, formerly Associate Professor of Anatomy, Peiping Union Medical College, China.

Francis M. Summers, University of California College of Agriculture.

The resignations were accepted with regret.

Members dropped for non-payment of dues for 2 years: It was explained that, while several members were in arrears for 2 years or more, no action is being taken because of the "moratorium during the war."

Reinstatement of member: It was voted that Howard B. Kellogg, whose membership lapsed in 1936, be reinstated on payment of arrears in dues.

Number of members: The Secretary presented the following statistics of membership:

Number of full members May 20, 1945	731	
Number of "provisional" members, May 20, 1945	79	
Total membership May 20, 1945		810
Deceased during year	6	
Resigned during year	3	9
		801
"Provisional" members voted to full membership, April 5, 1946	79	
New members elected April 5, 1946		33
Total full membership April 5, 1946		834

(1 reinstatement to be added when conditions are fulfilled.)

Payment to Local Committee: The Secretary explained that, in 1942, the sum of $250 had been allotted to the Local Committee, with the expectation that the amount would probably be greater in succeeding years. He further explained that the Executive Committee had voted that the Association should bear the expense of the Smoker, rather than to expect the host university to carry this expense. With this in mind the Executive Committee had suggested that $300 be appropriated for expenses of the meeting.

The Secretary then explained that new information indicated that $400 would be a more suitable amount; that the treasury could easily carry this sum. On motion duly made and seconded it was voted that the Treasurer be authorized to pay the sum of $400 from the general funds of the Association to the Local Committee on Arrangements, to aid in defraying the cost of entertainment (including the Smoker) of the Association during the present meeting.

Dues for 1946: The Secretary-Treasurer reported that the Executive Committee recommends that the dues for the year 1946 be $2.50. This is $0.50 above the pre-war level of $2.00, and is suggested in order to provide funds for increased clerical work and increased cost of meetings resulting in part from our enlarged membership. On motion duly made and seconded it was voted that the dues for 1946 be $2.50.

Place of next meeting: The President reported that the Executive Committee had not yet decided upon the place for the 1947 meeting. A meeting in the ''East'' is in order. He expressed the appreciation of the Association for an invitation from Professor Duncan to meet at the University of Texas, Galveston, and to Washington University, University of Illinois, Marquette University and the University of Wisconsin from each of which the Association has a standing invitation.

The Journals: The Secretary stated that the Executive Committee, which is authorized to act as Committee on Journals, had the following report:

On January 8, 1946, Philip E. Smith presented his resignation as Managing Editor of the American Journal of Anatomy, to take effect with the completion of the current volume, in May, 1946. Acting President Schaeffer appointed a special committee consisting of G. W. Corner, Chairman, E. J. Farris and P. E. Smith to nominate a successor. This committee decided unanimously upon Harold Cummins, Associate Editor, and this selection was confirmed by the Executive Committee, after the Executive Committee had voted to accept with regret the resignation of Philip E. Smith. The action is subject to the endorsement of The Wistar Institute, owner of the Journal.

In a letter dated January 10, 1946, George B. Wislocki presented his resignation as Associate Editor of The American Journal of Anatomy. His resignation was accepted with regret by the Executive Committee. This vacancy will be filled by the new Managing Editor.

By motion duly made and seconded, the action of the Executive Committee in the selection of Harold Cummins as the new Managing Editor was endorsed by the Association. The following motion was then presented by George W. Corner:

> Moved: that the Secretary express to Dr. Philip E. Smith the thanks of the Association for his effective service as Managing Editor of the American Journal of Anatomy during the past 5 years, and to Dr. George B. Wislocki for his services as Associate Editor for 9 years.

This motion was seconded and passed.

Regarding journals: For the benefit of new members it was pointed out that The American Journal of Anatomy and The Anatomical Record, while owned by The Wistar Institute, are sponsored by the American Association of Anatomists, which selects their Managing Editors subject to the approval of The Wistar Institute; and that all members of the Association are granted a reduction of 25% from the regular subscription rates, not only of those two journals but also of all The Wistar Journals.

Report of Representative to Union of American Biological Societies: E. V. Cowdry, who had been appointed by Acting President Schaeffer a Special Representative to the Union;

reported that at a meeting in St. Louis, on March 28, 1946, which was held by the Union in conjunction with several other biological societies, it was decided to request the adoption, by the individual societies in the field of biology, of a resolution which he proceeded to present to the meeting as follows:

> Resolved, that the American Association of Anatomists endorses the formation of an organization of biologists on the basis of the two following general propositions:
>
> 1. To provide a means for executing our public responsibilities as biologists and scientists, so that the freedom essential to the progress of science and public welfare be insured.
>
> 2. To safeguard the professional interests of biologists and to assist in providing the material means for the promotion of biological research; also to provide such services as may be necessary to facilitate this program.

On motion by Dr. Cowdry, duly seconded, this resolution was adopted by the meeting.

Question of incorporation and of accepting offer of Anatomical Journal Trust fund: The Secretary presented this matter as follows:

> The Trustees of the Anatomical Journal Trust Fund (Minot Memorial Fund) namely, G. W. Corner, Chairman, B. M. Patten and P. E. Smith, have offered to turn over to the American Association of Anatomists the fund totaling about $13,700, providing the Association agrees to utilize the funds for the declared purposes, and that the Association become incorporated. The Executive Committee recommends that the Association accept the fund, and that it incorporate. It further recommends that the newly elected President and Secretary-Treasurer be authorized to carry out the steps necessary for incorporation, and to prepare any necessary amendments to the Constitution, for presentation to the Association.
>
> The "declared purposes" of the fund, published in the Anatomical Record, 1936, v. 65, Supplement, pp. 13 and 14, are as follows: ". . . they hold the said funds for the purpose of collecting the income thereon and applying the income and so much of the principal as may be deemed by the Trustees expedient in the assistance of the publication of original investigations of anatomy and allied science, and for the purpose of publishing the Americal Journal of Anatomy and the Anatomical Record, should the same be turned back by The Wistar Institute to the Trustees."

Under authorization of the Executive Committee the Secretary-Treasurer has made preliminary studies of the matter of incorporation and has found that incorporation in the State of Delaware could be accomplished at a cost of $100, and maintained at an annual cost of $50.00.

On motion by the Secretary, duly seconded, it was voted that the Association accept the offer of the Trustees of the Anatomical Journal Trust Fund to turn over the fund to the American Association of Anatomists; that the Association become incorporated; and that the newly elected President and Secretary-Treasurer be authorized to proceed with the steps necessary for incorporation and to prepare amendments to the Constitution, if any are necessary, the latter to be brought before the Association in the prescribed manner.

Anatomical reviews: The Secretary reported receipt from one of the members of the Association of a letter advocating the formation by the Association of a new journal of anatomical reviews. He stated that the matter had been brought before the Executive Committee and that the Committee had voted to reaffirm action taken in 1938 when, after a year's study by a special committee of the same question, the decision had been made not to start such a journal.

A motion made and seconded that the Association endorse the action of the Executive Committee was passed by a standing vote of approximately 61 to 8.

Delegate to Celebration: For information it was reported that J. E. Markee had been appointed to represent the American Association of Anatomists at the final exercises of the Sesquicentennial Celebration of the University of North Carolina, Chapel Hill, N. C., April 12 and 13, 1946.

Other Anatomical Societies: For information, the Secretary reported regarding contacts with other anatomical societies that during the war, programs, abstracts and Proceedings have been interchanged with Dr. Cave, Secretary of the Anatomical Society of Great Britain and Ireland, that exchange has been impossible with the other six societies with which they had been planned; and that to the original seven has been added ''Sociedad de Anatomia Normal y Pathologica de Chile,'' Adolpho Escobar, Secretary.

Under call for other business, Dr. Cowdry moved that the Executive Committee consider the problem of improving the attractiveness of a career in anatomy. The motion was duly seconded and passed.

Professor R. M. Strong made the following motion: "I move that the Association express its appreciation of the conscientious and effective service of its Secretary-Treasurer, Professor Eliot R. Clark, during a period of 7 years." This motion was seconded and passed.

The following resolution was presented and adopted: moved, that the Association appreciates the able direction of its affairs during the 3 war years by Professor J. Parsons Schaeffer, who, elected First Vice-President, took over as Acting President and carried on without hesitation, after an almost simultaneous double blow, the deaths of President Edgar Allen and Secretary-Treasurer, Francis H. Swett.

Resolution: At the close of the Saturday afternoon session, the following resolution was presented by George W. Corner:

> The members of the American Association of Anatomists, at the fifty-ninth session held at Cleveland, April 4 to 6, 1946, express their grateful appreciation to the authorities of Western Reserve University and particularly to Dr. Normand Hoerr and the Local Committee for the highly efficient arrangements made for the meeting and for the generous hospitality which has made this session of the Association successful in every way.

This resolution was seconded and adopted unanimously and with enthusiasm.

Acting President Schaeffer then declared the Fifty-ninth Session at an end.

Eliot R. Clark

Secretary of the Fifty-ninth Session of
the American Association of Anatomists

AMERICAN ASSOCIATION OF ANATOMISTS

OFFICERS AND LIST OF MEMBERS

Officers

President .... George W. Corner
First Vice-President ....Edward A. Boyden
Second Vice-President ....Marion Hines
Secretary-Treasurer ....Norman L. Hoerr

Executive Committee

For term expiring 1947 ....Joseph C. Hinsey, Carl C. Speidel
For term expiring 1948 ....E. Horne Craigie, Walter E. Sullivan
For term expiring 1949 ....Sam L. Clark, S. I. Kornhauser
For term expiring 1950 ....William Bloom, William W. Greulich

Delegates to the Council of A.A.A.S. for 1946

John L. Bremer
Frederic T. Lewis

Representative to the National Research Council 1946–1949

Carl C. Speidel

Member of the Commission on Standardization of Biological Stains

H. A. Davenport

Delegate to Union of Biological Societies, 1946–1949

Stacy R. Guild

Committee on Nominations for 1947

Bradley M. Patten, *Chairman*
Davenport Hooker
Eben J. Carey

Committee on Anatomical Nomenclature

George W. Corner, *Chairman*
Edward A. Boyden
C. H. Danforth
Sam L. Clark
W. W. Greulich

Motion Picture Committee

Eliot R. Clark, *Chairman*
Warren H. Lewis
Carl C. Speidel

MEMBERS

Abell, Richard G., M.A., Ph.D., Assistant Professor of Anatomy, *University of Pennsylvania School of Medicine, Philadelphia 4, Pa.*

Abercrombie, Warren F., A.B., Sc.M., Ph.D., Head of Biology Department, *Erskine College, Due West, S. C.*

Aberle, (mrs.) Sophie D., Ph.D., M.D., *National Research Council, 2101 Constitution Ave., Washington, D. C.*

Adams, A. Elizabeth, A.B., A.M., Ph.D., Professor of Zoology, *Mount Holyoke College, South Hadley, Mass.*

Adams, L. A., A.B., A.M., Ph.D., Professor of Zoology, Curator of Museum of Natural History, University of Illinois, *401 Vermont St., Urbana, Ill.*

Adamstone, F. B., B.A., M.A., Ph.D., Associate Professor of Zoology, *345 Natural History Bldg., University of Illinois, Urbana, Ill.*

Addison, William Henry Fitzgerald, B.A., M.D., Professor of Normal Histology and Embryology, *School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.*

Adelmann, Howard B., B.A., M.A., Ph.D., Professor of Histology and Embryology and Chairman, Department of Zoology, *Cornell University, Ithaca, N. Y.*

Ades, Harlow W., B.S., M.S., Ph.D., Assistant Professor of Anatomy, *Box 759, Emory University, Emory, Ga.*

Alden, Roland H., B.A., Ph.D., Assistant Professor of Anatomy, *University of Tennessee College of Medicine, Memphis, Tenn.*

Alexander, William F., B.S., M.S., Ph.D., Assistant Professor of Microanatomy, *School of Medicine, St. Louis University, 1402 S. Grand Ave., St. Louis 4, Mo.*

Algire, Glenn H., M.D., Assistant Surgeon (R), U.S.P.H.S., *National Cancer Institute, Bethesda, Md.*

Allen, Bennet Mills, Ph.B., Ph.D., D.S. (honorary), Professor of Zoology, University of California at Los Angeles, *147 N. Bowling Green Way, Los Angeles 24, Calif.*

Allen, Ezra, A.M., Ph.D., Retired. Visiting Professor, J. B. Stetson University, *237 W. Stetson St., De Land, Fla.*

Allen, Lane, Ph.D., M.D., Professor of Anatomy, *University of Georgia, School of Medicine, Augusta, Ga.*

Allen, Willard M., B.S., M.S., M.D., *Washington University, School of Medicine, St. Louis, Mo.*

Allen, William F., A.M., Ph.D., D.Sc., honoris causa, Head of Department of Anatomy, *University of Oregon Medical School, Portland, Ore.*

Allis, Edward Phelps, Jr., C.E., M.D., LL.D. (Charter member), *Palais de Carnolès, Menton (A.M.), France.*

Altschul, Rudolf, M.D., Associate Professor in Histology and Neurology, *University of Saskatchewan, Saskatoon, Canada.*

Andersen, Dorothy H., Associate in Pathology, Columbia University; Assistant Pathologist, *Babies' Hospital, 167th St. and Broadway, New York City.*

Andrew, Warren, M.S., M.D., Ph.D., Associate Professor of Histology and Embryology, *Department of Anatomy, Southwestern Medical College, Dallas, Tex.*

Andrews, Frederick N., B.S., M.S., Ph.D., Associate Professor of Animal Husbandry, *Department of Animal Husbandry, Purdue University, Lafayette, Ind.*

Angel, John L., A.B., Ph.D., Associate in Anatomy and Physical Anthropology, *Jefferson Medical College, Daniel Baugh Institute of Anatomy, 307 S. Eleventh St., Philadelphia, Pa.*

Angulo y Gonzalez, Armando W., B.L., B.S., A.B., A.M., Ph.D., *418 Beverly Blvd., Upper Darby, Pa.*

Anson, Barry Joseph, A.B., A.M., Ph.D., Professor of Anatomy, *Northwestern University Medical School, 303 East Chicago Ave., Chicago, Ill.*

Apgar, Bessie D., Ph.D., *Elizabethtown College, Elizabethtown, Pa.*

Apgar, Charles S., Jr., Ph.D., *Elizabethtown College, Elizabethtown, Pa.*

Appleby, J. I., A.B., M.B., A.M., M.D., Physician and Surgeon, Company Surgeon, N. Y. C. & St. L. R. R. Co., *105 1/2 East Main St., Bellevue, O.*

Arey, Leslie B., Ph.D., Sc.D., (Ex Com. '30–'34, Second Vice-Pres. '42–), Robert Laughlin Rea Professor of Anatomy, and Chairman of the Division of Anatomy, *Northwestern University Medical School, 303 East Chicago Ave., Chicago, Ill.*

Armstrong, Philip Brownell, B.S., M.D., Professor of Anatomy, *College of Medicine, Syracuse University, Syracuse, N. Y.*

Asdell, Sydney Arthur, M.A., Ph.D., Professor of Animal Physiology, State College of Agriculture, *Cornell University, Ithaca, N. Y.*

Ashley, Laurence M., B.S., A.M., Ph.D., Instructor in Microscopic Anatomy, *College of Medical Evangelists, Loma Linda, California.*

Atkinson, William Brockliss, B.S., M.S., Ph.D., Instructor in Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32, N. Y.*

Atterbury, Ruth Rand, A.M., Ph.D., *R.F.D. 1, Roslyn, L. I., N. Y.*

Bacon, Robert Lewis, B.S., M.S., Ph.D., Instructor in Anatomy, *Department of Anatomy, Stanford University, California.*

Badertscher, Jacob A., Ph.D., Professor of Anatomy, Indiana University School of Medicine, *312 South Fess Ave., Bloomington, Ind.*

Bagley, Charles, Jr., M.D., Professor of Neurosurgery, University of Maryland Medical School; Associate in Experimental Neurology, Johns Hopkins University Medical School, *The Latrobe Apts., Baltimore, Md.*

Bailey, Percival, M.D., Ph.D., Professor of Neurology and Neurological Surgery, *University of Illinois, 912 South Wood St., Chicago, Ill.*

Bailey, Ralph Jordan, B.S., M.S., Ph.D. (Address unknown; information desired).

Bailliff, R. N., B.A., M.A., Ph.D., Assistant Professor of Anatomy, *Louisiana State University Medical Center, New Orleans 13, La.*

Baitsell, George Alfred, B.S., M.A., Ph.D., Professor of Biology, Yale University, *Osborn Zoological Laboratory, New Haven, Conn.*

Baker, Burton Lowell, M.S., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, University of Michigan, Ann Arbor, Mich.*

Baker, Dan Dysart, A.B., M.A., M.D., Orthopaedic Surgery, *15th floor, Hibernia Bank Bldg., New Orleans 12, La.*

BAKER, R. C., A.B., M.A., Ph.D., Professor of Anatomy, *Department of Anatomy, Hamilton Hall, Ohio State University, Columbus, O.*

BALDWIN, WESLEY MANNING, A.B., A.M., M.D., *Northfield, Minnesota.*

BARDEN, ROBERT BRADSHAW, A.B., Ph.D., Instructor in Anatomy, *University of Southern California, School of Medicine, Los Angeles 7, Calif.*

BARNARD, JOHN W., M.S., Ph.D., Assistant Professor of Anatomy, *School of Medicine, University of Oklahoma, Oklahoma City, Okla.*

BARRETT, W. C., JR., Ph.D., Assistant Professor of Histology and Embryology, *School of Medicine, Western Reserve University, Cleveland, O.*

BARRIS, RALPH W., Ph.D., (address unknown; information desired).

BARRON, DONALD H., B.A., M.S., Ph.D., Associate Professor of Physiology, *Yale University School of Medicine, New Haven, Conn.*

BARRY, ALEXANDER, A.B., M.A., Ph.D., Instructor in Anatomy, University of Michigan Medical School, *1430 Golden Ave., Ann Arbor, Mich.*

BARTELMEZ, GEORGE W., Ph.D. (Vice-Pres. '32–'34, Exec. Com. '36–'40). Professor of Anatomy, *The University of Chicago, Chicago 37, Ill.*

BARTSCH, PAUL, B.S., M.S., Ph.D., D.Sc., Curator of Mollusks and Cenozoic Invertebrates, *U. S. National Museum, Washington, D. C.*

BASSETT, DAVID L., A.B., M.D., Assistant Professor of Anatomy, *Stanford University School of Medicine, Stanford University, Calif.*

BAST, T. H., B.A., Ph.D., Professor of Anatomy, *University of Wisconsin, 308 Science Hall, Madison, Wis.*

BATSON, O. V., A.M., M.D., Professor of Anatomy, *Department of Anatomy, Graduate School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.*

BAUMGARTNER, WILLIAM J., A.B., A.M., Ph.D., Professor of Zoology, University of Kansas, *1209 Ohio St., Lawrence, Kan.*

BEAMS, HAROLD W., Ph.D., Professor of Zoology, *State University of Iowa, Iowa City, Ia.*

BEATON, LINDSAY E., A.B., M.S., M.D., Medical Fellow, National Research Council, *Northwestern University Medical School, Chicago, Ill.*

BEATTIE, JOHN, M.D., D.Sc., Conservator of Museum and Director of Research, Royal College of Surgeons; Colonel, Army Medical Service; *Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2, England.*

BECKER, R. FREDERICK, B.S., M.S., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, School of Medicine, University of Washington, Seattle 5, Wash.*

BÉLANGER, LEONARD F., A.B., M.D., A.M., Assistant Professor of Histology and Embryology, *Department of Anatomy, McGill University, Montreal, Canada.*

BENJAMIN, JAMES W., A.B., M.S., Ph.D., Associate Professor of Histology, Embryology and Neuroanatomy, *New York Medical College, Flower and Fifth Avenue Hospitals, New York City.*

BENNETT, GEORGE ALLEN, A.B., M.D., Associate Professor of Anatomy, *Daniel Baugh Institute of Anatomy, Jefferson Medical College, Philadelphia, Pa.*

BENNETT, HENRY STANLEY, A.B., M.D., Assistant Professor of Cytology, *Department of Biology, Massachusetts Institute of Technology, Cambridge, Mass.*

BENOIT, JACQUES, M.D., Professeur d'Embryologie, *Institut d'Embryologie, Faculté de Medecine, Strasbourg, France.*

Bensley, Robert Russell, A.B., M.B., Sc.D. (Second Vice-Pres. '06–'07, Ex. Com. '08–'12, President '18–'20), Professor Emeritus of Anatomy, *The University of Chicago, Chicago, Ill.*

Bensley, Sylvia H., A.B., M.D., Assistant Professor of Anatomy, *Department of Anatomy, The University of Chicago, Chicago, Ill.*

Benton, Joseph G., A.B., M.Sc., Ph.D., M.D., Research Fellow, Department of Pharmacology, *Cornell University Medical College, 1300 York Avenue, New York 21, N. Y.*

Bergmann, Louis Lawrence, M.D., Instructor in Anatomy, *New York University College of Medicine, 477 First Avenue, New York 16, N. Y.*

Berry, Charles M., A.B., M.S., Ph.D., Instructor in Anatomy, *Cornell University Medical College, 1300 York Ave., New York City.*

Best, R. Russell, B.Sc., M.D., Assistant Professor of Anatomy, Associate Professor of Surgery, University of Nebraska, College of Medicine, *527 Medical Arts Building, Omaha, Neb.*

Bevelander, Gerrit, Ph.D., Associate Professor of Anatomy, *New York University, 477 First Ave., New York City.*

Bigelow, Robert P., S.B., Ph.D., Professor Emeritus of Zoology and Parasitology, Massachusetts Institute of Technology, *72 Blake Rd., Brookline, Mass.*

Bishop, Katherine Scott, A.B., M.D., *1508 La Loma Ave., Berkeley, Calif.*

Blair, Duncan M., M.B., Ch.B., D.Sc., Professor of Anatomy, *University of Glasgow, Glasgow, Scotland.*

Blair, Vilray Papin, M.D., A.B., Professor of Clinical Surgery, Washington University School of Medicine, *400 Metropolitan Building, St. Louis, Mo.*

Blaisdell, Frank Ellsworth, Sr., M.D., Emeritus Professor of Surgery, Medical Department of Stanford University, *1900 Washington St., San Francisco, Calif.*

Blandau, Richard J., A.B., Ph.D., Instructor in Anatomy, *School of Medicine and Dentistry, University of Rochester, Rochester, New York.*

Blincoe, Homer, A.B., M.S., M.D., Professor of Anatomy, *Emory University, Emory, Ga.*

Bloom, William, A.B., M.D., Professor of Anatomy, *Department of Anatomy, The University of Chicago, Chicago, Ill.*

Blount, Isabel H., A.B., A.M., Ph.D., Voluntary Research Worker, *Department of Anatomy, University of Texas, Medical School, Galveston, Tex.*

Blount, Raymond Frank, M.S., Ph.D., Professor of Anatomy, *Department of Anatomy, University of Texas, Medical Branch, Galveston, Tex.*

Blumenfeld, Charles M., Ph.D., M.D., Pathologist, Sutter Hospital, *1433 41st Street, Sacramento 16, California.*

Bodian, David, S.B., Ph.D., M.D., Assistant Professor of Epidemiology, *Johns Hopkins University School of Hygiene and Public Health, Poliomyelitis Laboratory, 1901 E. Madison Street, Baltimore 5, Md.*

Bogart, Ralph, B.S., M.S., Ph.D., Agent in Animal Husbandry, Bureau of Animal Industry, U. S. D. A., Instructor in Animal Husbandry, *University of Missouri, 324 Mumford Hall, Columbia, Mo.*

BOLING, JOHN L., A.B., Ph.D., Instructor in Anatomy, *College of Medicine, New York University, 477 First Ave., New York City 16.*

BOOK, M. HAROLD, M.D., B.Sc. (Med.), Director of Laboratories, *Norristown State Hospital, Norristown, Pa.*

BOWIE, DONALD J., B.Sc., Med., M.A., Ph.D., Assistant Professor of Anatomy, *Medical College, Winnipeg, Manitoba, Canada.*

BOYD, EDITH, M.D., Superintendent of Whitefish Hospital, *Whitefish, Mont.*

BOYDEN, EDWARD ALLEN, A.M., Ph.D. (Ex. Com. '28–'32, '39–'46, 1st Vice-Pres. '46–), Professor of Anatomy and Chairman of Department, *Institute of Anatomy, University of Minnesota, Minneapolis, Minn.*

BOYER, ESTHER L., M.A., Ph.D., M.D., *Western Montana Clinic, Missoula, Mont.*

BRADBURY, JAMES T., B.S., M.S., Sc.D., *Dept. of Obstetrics and Gynecology, University Hospitals, Iowa City, Iowa.*

BRADLEY, E. MORTON, B.A., Ph.D., *Temple University School of Medicine, Broad St. at Ontario, Philadelphia 40, Pa.*

BRASHEAR, ALTON D., A.B., B.S., M.S., D.D.S., F.A.C.D., Associate Professor of Anatomy, *Department of Anatomy, Medical College of Virginia, Richmond 19, Va.*

BREMER, JOHN LEWIS, A.B., M.D. (Ex Com. '15–'18, '33–'37), Hersean Professor of Anatomy Emeritus, *Harvard Medical School, Boston, Mass.*

BREWER, JOHN I., S.B., M.D., Ph.D., Associate in Gynecology, Northwestern University Medical School and St. Luke's Hospital, Chicago, *104 S. Michigan, Chicago, Ill.*

BRODIE, ALLAN G., D.D.S., Ph.D., Professor of Orthodontia and Head of the Department, *College of Dentistry, University of Illinois, 30 N. Michigan Ave., Chicago, Ill.*

BROWMAN, LUDWIG G., Ph.D., Associate Professor in Zoology and Physiology, *Montana State University, Missoula, Mont.*

BROWN, JAMES O., B.S., M.S., Ph.D., Associate Professor of Anatomy, *Woman's Medical College of Pennsylvania, Philadelphia, Pa.*

BRUES, AUSTIN MOORE, A.B., M.D., *Department of Medicine, University of Chicago, Chicago, Illinois.*

BRUESCH, SIMON RULIN, A.B., M.S., M.B., Ph.D., M.D., Instructor in Anatomy, *University of Tennessee College of Medicine, Memphis, Tenn.*

BRYCE, THOMAS H., M.A., M.D., F.R.S., Emeritus Professor of Anatomy, University of Glasgow, *The Loaning, Peebles, Scotland.*

BUCHANAN, A. R., B.S., M.S., M.D., Associate Professor of Anatomy, *University of Colorado Medical School, 4200 E. Ninth Ave., Denver, Colo.*

BUEKER, ELMER D., M.A., Ph.D., Assistant Professor of Anatomy, *Medical College of the State of South Carolina, Charleston, S. C.*

BULLARD, H. HAYS, A.M., Ph.D., M.D., *18 Tracy St., Rochester 7, N. Y.*

BULLOCK, THEODORE HOLMES, Ph.D., Assistant Professor of Anatomy, *University of Missouri School of Medicine, Columbia, Missouri.*

BUNTING, CHARLES HENRY, B.S., M.D., Professor of Pathology, *139 Armory Street, Hamden, Connecticut.*

BURACK, ETHEL, Ph.D., M.D., Assistant Professor of Anatomy, *Albany Medical College, Albany, N. Y.*

Burns, B. I., A.B., A.M., Ph.D., M.D., Medical Director of Hospitals, *University of Texas, Medical Branch, Galveston, Texas.*

Burns, Robert K., Jr., A.B., Ph.D., Research Associate, Department of Embryology, *Carnegie Institution of Washington, Wolfe and Madison Sts. Baltimore, Md.*

Burr, Harold Saxton, Ph.D., (Ex. Com. '29–'33), E. K. Hunt Professor of Anatomy, Head of Section of Neuro-Anatomy, School of Medicine, Yale University, *333 Cedar St., New Haven, Conn.*

Burrows, Montrose T., A.B., M.D., Physician and Surgeon, *201 North El Malino Ave., Pasadena, Calif.*

Butcher, Earl O., A.B., M.A., Ph.D., Associate Professor of Anatomy, *New York University, 477 First Ave., New York City.*

Butler, Elmer G., A.M., Ph.D., Sc.D., Professor of Zoology, *Department of Biology, Princeton University, Princeton, N. J.*

Calkins, Leroy A., M.D., M.S., Ph.D., Professor of Obstetrics and Gynecology, *University of Kansas Hospital, Kansas City, Kan.*

Cameron, Angus L., A.B., M.S., M.D., Ph.D., *Northwest Clinic, Minot, N. Dak.*

Cameron, John A., A.B., A.M., Ph.D., Professor of Anatomy and Chairman of the Department, *Baylor University, College of Dentistry, Dallas 1, Tex.*

Campbell, Berry, A.B., Ph.D., Assistant Professor of Anatomy, *Medical School, University of Minnesota, Minneapolis, Minn.*

Candela, Pompeo B., M.D., Research Associate in Anatomy, New York Medical College, *576 18th St., Brooklyn, N. Y.*

Carey, Eben J., M.S., Sc.D., M.D., Dean and Professor of Anatomy and Director of Anatomy Department, Marquette University School of Medicine, *6119 W. Wisconsin Ave., Wauwatosa, Wis.*

Carpenter, Esther, B.A., M.S., Ph.D., Associate Professor of Zoology, *Smith College, Northampton, Mass.*

Carpenter, Russell L., B.S., Ph.D., Professor of Zoology, *Tufts College, Medford, Mass.*

Carver, Gail L., A.B., A.M., Professor of Biology, *Mercer University, Macon, Ga.*

Casamajor, Louis, A.M., M.D., Professor of Clinical Neurology, *Columbia University, 706 West 168th St., New York City.*

Casey, Albert E., M.D., Pathologist, Baptist, Children's, Holy Name of Jesus and Jefferson Hospitals, *1907 Wellington Rd., Birmingham, Ala.*

Castelnuovo, Gina, Ph.D., Assistant Professor of Anatomy, *Woman's Medical College of Pennsylvania, Philadelphia, Pa.*

Chambers, Robert, Jr., M.A., Ph.D., L.L.D., (Second Vice-Pres. '39–'40), Research Professor of Biology, *New York University, New York City.*

Chandler, Simon B., A.B., A.M., M.D., Professor of Anatomy, *West Virginia University School of Medicine, Morgantown, W. Va.*

Chang, Chun, M.D., Associate Professor of Anatomy, *National Medical College of Shanghai, 373 Ave. Haig, Shanghai, China.*

Charipper, Harry A., B.S., M.S., Ph.D., Professor of Biology (Tissue Cytology, Endocrinology, Histology and Comparative Haematology), Chairman of Department, *Washington Square College, New York University, New York City.*

Charles, Cecil M., A.B., M.A., Ph.D., M.D., Associate Professor of Anatomy, *Washington University School of Medicine, St. Louis, Mo.*

Chase, Philena Evans, B.A., Ph.D., with the American Red Cross for the duration. *56 Leland St., Portland, Me.*

Chase, Ralph E., A.B., B.Sc., M.A., Instructor in Anatomy, *Oklahoma University Medical School, Oklahoma City, Okla.*

Chase, Samuel Wood, A.B., A.M., Ph.D., Associate Professor of Histology and Embryology, *Western Reserve University, 2109 Adelbert Road, Cleveland 6, O.*

Cherner, Maxwell, M.D., Police and Fire Surgeon, Dept. of Public Safety, Phila., Medical Examiner, Reading Railroad, *34 W. Phil-Ellena Street, Philadelphia 19, Pa.*

Child, Charles Manning, M.S., Ph.D., D.Sc., Professor Emeritus of Zoology, *Jordan Hall, Stanford University, Calif.*

Chouke, Kehar S., M.A., M.D., Assistant Professor of Anatomy, *Graduate School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.*

Christensen, Kermit, Ph.D., Associate Professor of Microanatomy, *St. Louis University School of Medicine, 1402 South Grand Blvd., St. Louis, Mo.*

Clark, Eleanor Linton, A.M., Voluntary Research Worker, Department of Anatomy, *Medical Department, University of Pennsylvania, Philadelphia 4, Pa.*

Clark, Eliot R., A.B., M.D., Sc.D., (Ex. Com. '16–'19, Sec.-Treas. '38–'42; Sec.-Treas. '43–'46), Professor of Anatomy and Director of Department of Anatomy, *Medical Department, University of Pennsylvania, Philadelphia 4, Pa.*

Clark, George, Ph.D., Assistant Professor of Psychobiology, *Yerkes Laboratories of Primate Biology, Orange Park, Fla.*

Clark, Sam L., M.S., Ph.D., M.D., (Ex. Com. '42–) Professor of Anatomy, *Vanderbilt University School of Medicine, Nashville, Tenn.*

Clausen, Harry J., A.B., M.S., Ph.D., Professor of Anatomy, School of Dentistry, Loyola University, *6363 St. Charles Ave., New Orleans, La.*

Clements, Leo, Ph.D., Associate Professor of Anatomy, Member Nebraska Basic Science Board, *Creighton University Medical School, Omaha, Neb.*

Cleveland, Rucker, Ph.D., Instructor, Institutum Divi Thomae; medical writer (free lance), *188 E. McMillan, Cincinnati 19, Ohio.*

Cobb, W. Montague, A.B., M.D., Ph.D., Professor of Anatomy, *Howard University, 520 W. St., N. W., Washington, D. C.*

Coe, Wesley R., Ph.D., Professor of Biology Emeritus, *Yale University, Osborn Zoological Laboratory, New Haven, Conn.*

Cohn, Alfred E., A.B., M.D., Member, *Rockefeller Institute for Medical Research, New York City, N. Y.*

Cole, Arch Evan, B.A., Ph.D., Associate Professor of Anatomy, *University of Louisville; School of Medicine, 101 W. Chestnut St., Louisville, Ky.*

Cole, Elbert C., Ph.D., Professor of Biology, *Williams College, Williamstown, Mass.*

Cole, Harold H., Ph.D., Professor of Animal Husbandry, University of California, and Animal Husbandman, *Experimental Station of the College of Agriculture, Davis, Calif.*

COLLINGS, M. RAYMOND, A.B., M.D., Professor of Anatomy, Medical College, Wayne University, *1512 St. Antoine St., Detroit, Mich.*

COMAN, DANA, A.B., A.M., M.D., *145 West 57th St., New York City 19.*

CONEL, JESSE LEROY, A.M., Ph.D., Professor of Anatomy, Boston University School of Medicine, *45 Arlington St., Newton, Mass.*

CONGDON, EDGAR DAVIDSON, Ph.D., Professor of Anatomy, Department of Anatomy, Chicago Medical School, *211 N. Oak Park Ave., Oak Park, Illinois.*

CONKLIN, EDWIN GRANT, A.M., Ph.D., Sc.D., L.L.D., Professor Emeritus of Biology, Princeton University, *139 Broadmead Ave., Princeton, N. J.*

COPENHAVER, WILFRED M., A.B., Ph.D., Associate Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 632 West 168th St., New York City.*

CORBIN, KENDALL B., A.B., M.D., (After October 1, 1946) Consulting Physician, *Section on Neurology, Mayo Clinic, Rochester, Minn.*

COREY, EDWARD LYMAN, A.B., Ph.D., Assistant Professor of Physiology, *University of Virginia School of Medicine, University, Va.*

CORNER, GEORGE W., A.B., M.D., D.Sc., (Ex. Com. '26–'30, '38–'42; Sec.-Treas. '30–'38; Pres. '46–), Director, Department of Embryology, *Carnegie Institution of Washington, Wolfe and Madison Sts., Baltimore 5, Md.*

CORNING, H. K., M.D., Professor of Anatomy (retired), *1107 Fifth Ave., New York City.*

COUNT, EARL W., A.B., B.D., Ph.D., Research Associate, *The Viking Fund, Inc., 14 East 71st St., New York 21, N. Y.*

COVELL, W. P., M.S., Ph.D., M.D., Lecturer in Otology, Rhinology and Laryngology, University of California Medical Center; Associate Professor of Research Medicine, *George Williams Hooper Foundation, San Francisco, Calif.*

COWDRY, EDMUND V., Ph.D. (Ex. Com. '26–'30, 1st Vice-Pres. '34–'36), Professor of Anatomy, *Washington University School of Medicine, St. Louis, Mo.*

CRAFTS, ROGER CONANT, B.S., Ph.D., Assistant Professor of Anatomy, *Boston University School of Medicine, 80 E. Concord St., Boston, Mass.*

CRAIGIE, E. HORNE, B.A., Ph.D. (Ex. Com. '41–), Professor of Comparative Anatomy and Neurology, *Department of Zoology, University of Toronto, Toronto 5, Canada.*

CROSBY, ELIZABETH CAROLINE, M.S., Ph.D., Professor of Anatomy, University of Michigan, *Department of Anatomy, University of Michigan, Ann Arbor, Mich.*

CROUCH, RICHARD L., Ph.D., M.D., Associate Professor of Anatomy, *University of Missouri, Columbia, Mo.*

CUAJUNCO, FIDEL, M.D., Associate Professor of Anatomy, College of Medicine, University of the Philippines, *620 Colorado St., Manila, P. I.*

CULLEN, THOMAS S., M.B., D.Sc., LL.D. (Tor.), Emeritus Professor of Gynecology, Johns Hopkins University, Visiting Gynecologist to the Johns Hopkins Hospital, *20 E. Eager St., Baltimore, Md.*

CUMMINS, HAROLD, A.B., Ph.D. (Ex. Com. '36–'40), Professor of Anatomy and Head of Department of Anatomy, *Tulane University Medical School, New Orleans 15, La.*

CUNNINGHAM, ROBERT S., B.S., A.M., M.D., Sc.D. (Ex. Com. '32–'36), Professor of Anatomy and Dean, Albany Medical College; Administrator, Albany Hospital, *47 New Scotland Ave., Albany 3, N. Y.*

CURTIS, GEORGE M., A.M., Ph.D., M.D., Professor of Surgery and Chairman of the Department of Research Surgery; *Kinsman Hall, Ohio State University, Columbus, O.*

CURWEN, ALICE O., Ph.D. (Mrs. Edward F. McKeen), *524 French Street, Bangor, Maine.*

CUTULY, EUGENE, B.S., M.S., Ph.D., Pfc. U. S. Army, *528 Halcomb Avenue, Clairton, Pa.*

DALTON, ALBERT J., B.S., M.A., Ph.D., Senior Cytologist, *National Cancer Institute, National Institute of Health, Bethesda, Md.*

DANFORTH, CHARLES HASKELL, A.B., A.M., Ph.D., Sc.D., (Ex. Com. '27–'31, Second Vice-Pres. '36–'38), Professor of Anatomy, *Stanford University, Stanford University, Calif.*

D'ANGELO, SAVINO ALBERT, B.A., M.S., Ph.D., Instructor in Biology, Washington Square College, New York University, *151-19-34 Ave., Flushing, N. Y.*

DARDINSKI, VINCENT J., A.B., M.A., M.D., Ph.D., Associate Professor of Pathology, Georgetown Medical School; Director of Laboratories, Georgetown University Hospital, *Georgetown University Hospital, Washington, D. C.*

DARON, GARMAN H., B.S., Ph.D., Associate Professor of Anatomy, *Georgetown University, School of Medicine, Washington, D. C.*

DARRACH, WILLIAM, A.B., A.M., M.D., Sc.D., LL.D., Professor of Clinical Surgery and Dean Emeritus of Faculty of Medicine, College of Physicians and Surgeons, Columbia University, *180 Fort Washington Ave., New York City.*

DAVENPORT, H. A., M.D., Associate Professor of Anatomy, *Northwestern University Medical School, Chicago, Ill.*

DAVIS, CARL L., M.D., Professor of Anatomy, University of Maryland School of Medicine, *Lombard and Greene Sts., Baltimore, Md.*

DAVIS, WARREN B., M.D., D.Sc., F.A.C.S., Clinical Professor of Oral Surgery, Jefferson Medical College; Maxillo-Facial Surgeon, Jefferson Hospital; Consulting Maxillo-Facial Surgeon, Kensington Hospital and Frankford Hospital; *135 S. 18th St., Philadelphia, Pa.*

DAWSON, ALDEN B., A.B., Ph.D., D.Sc., Professor of Zoology, *Biological Laboratories, Harvard University, Cambridge 38, Mass.*

DAWSON, HELEN L., B.S., M.S., Ph.D., Assistant Professor of Anatomy, *The State University of Iowa, College of Medicine, Iowa City, Ia.*

DAY, CAMERON D., Ph.D., Professor of Zoology, *Westminster College, Fulton, Mo.*

DEANE, HELEN WENDLER, B.A., M.A., Ph.D., Instructor in Anatomy, *Harvard Medical School, Boston, Mass.*

DE BRUYN, PETER P. H., M.D., Assistant Professor of Anatomy, *University of Chicago, Chicago 37, Ill.*

DECARLO, JOHN, M.D., Associate in Topographic and Applied Anatomy, Jefferson Medical College, *1629 S. Broad St., Philadelphia, Pa.*

DEES-MATTINGLY, MARIE, A.B., B.S., M.D., Associate Professor of Anatomy, *Tulane University of Louisiana, New Orleans, La.*

DE GARIS, CHARLES F., M.D., Ph.D., F.A.C.P., Professor of Anatomy, *University of Oklahoma, School of Medicine, Oklahoma City, Okla.*

DEIGNAN, STELLA L., B.S., M.S., Ph.D., Consultant, Work Projects Administration, *2236 Decatur Place, N. W., Washington 8, D. C.*

DEMPSEY, EDWARD W., Ph.D., Assistant Professor of Anatomy, *Harvard Medical School, 25 Shattuck St., Boston, Mass.*

DEMPSTER, WILFRID TAYLOR, Sc.D., Assistant Professor of Anatomy, *University of Michigan Medical School, Ann Arbor, Mich.*

DERBYSHIRE, ARTHUR J., Ph.D., Assistant Professor of Physiology, *Wayne University School of Medicine, 1512 St. Antoine St., Detroit, Mich.*

DE ROBERTIS, EDUARDO D. P., Member of Staff of Institute of General Anatomy, *University of Buenos Aires, Faculty of Medicine, Cangallo 2447, Bueons Aires, Argentina.*

DETWILER, SAMUEL RANDALL, Ph.B., A.M., Ph.D., M.S. (Hon.), (Ex. Com. '30–'34), Professor of Anatomy, Executive Officer of the Department, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York 32, N. Y.*

DEY, FREDERICK L., A.B., M.S., Ph.D., Research Fellow, Institute of Neurology, *Northwestern University Medical School, 303 East Chicago Ave., Chicago, Ill.*

DOAN, CHARLES A., M.D., B.S., Dean, College of Medicine, and Director of Medical Research, *Ohio State University, Columbus, O.*

DODDS, GIDEON S., M.A., Ph.D., Professor of Histology and Embryology, *West Virginia University Medical School, Morgantown, W. Va.*

DOMM, L. V., A.B., Ph.D., Associate Professor of Zoology, *Whitman Laboratory of Experimental Zoology, The University of Chicago, Chicago 37, Ill.*

DONALDSON, JOHN C., Ph.B., M.D., Professor of Anatomy, *School of Medicine, University of Pittsburgh, Pittsburgh, Pa.*

DOUGHERTY, THOMAS F., Ph.D., Instructor, Department of Anatomy, *Yale University, 333 Cedar St., New Haven, Conn.*

DOW, ROBERT S., A.B., M.A., M.D., Ph.D., Associate Professor of Anatomy and Clinical Instructor in Neurology, *University of Oregon Medical School, Portland, Ore.*

DOWNEY, HAL, A.M., Ph.D., Professor of Anatomy, *University of Minnesota Medical School, Minneapolis, Minn.*

DOYLE, WILLIAM LEWIS, M.A., Ph.D., Associate Professor of Anatomy, *Hull Anatomical Laboratory, University of Chicago, Chicago 37, Ill.*

DRAGER, GLENN A., B.S., M.S., Ph.D., Assistant Professor of Neuroanatomy, Dept. of Anatomy, *University of Texas, Medical Branch, Galveston, Tex.*

DUBOIS, FRANKLIN S., A.B., M.S., M.D., Assistant Medical Director, Silver Hill Foundation, *Silver Hill, New Canaan, Conn.*

DUESBERG, JULES, M.D., Professor of Anatomy, *University of Liege, 22 quai Mativa, Liege, Belgium.*

DUKE, KENNETH L., A.B., Ph.D., Associate in Anatomy, *Duke University School of Medicine, Durham, N. C.*

DUNCAN, DONALD, B.A., M.A., Ph.D., Professor of Anatomy, and Chairman of the Department, *School of Medicine, University of Texas, Galveston, Texas.*

DUSHANE, GRAHAM PHILLIPS, Ph.D., Associate Professor of Zoology, *The University of Chicago, Chicago 37, Ill.*

EARLE, WILTON R., Ph.D., Principal Cytologist, *National Cancer Institute, Bethesda, Md.*

Eastlick, Herbert L., A.B., M.S., Ph.D., Associate Professor of Zoology, *Washington State College, Pullman, Wash.*

Edds, Mac Vincent, Jr., B.A., M.A., Ph.D., Instructor in Anatomy, *Department of Anatomy, University of Pittsburgh, Pittsburgh 13, Pa.*

Edwards, Edward A., M.D., Surgeon, Fairview Hospital, *29 Lewis Ave., Great Barrington, Mass.*

Edwards, J. Graham, M.A., Ph.D., Associate Professor of Anatomy, University of Buffalo School of Medicine, *24 High St., Buffalo, N. Y.*

Edwards, Linden F., B.A., M.S., Ph.D., Professor of Anatomy, Ohio State University College of Medicine, *Hamilton Hall, Columbus 10, O.*

Elftman, Alice G., A.B., A.M., Ph.D., Instructor in Biological Sciences, *Hunter College, New York City.*

Elftman, Herbert, Ph.D., Assistant Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York City.*

Elias, Michael Hans, Ph.D., Information Specialist, U. S. Public Health Service, *605 Volunteer Bldg., Atlanta 3, Ga.*

Elliott, Harry Chandler, A.B., M.A., Ph.D., Research Fellow, U. S. Research Council, *Department of Anatomy, University of Toronto, Toronto, Canada.*

Elliott, Rush, A.B., A.M., Ph.D., Professor of Anatomy, Department of Zoology, Ohio University, *3 Marietta Ave., Athens, O.*

Elwyn, Adolph, A.M., Associate Professor of Neuroanatomy, *Columbia University, 630 West 168th St., New York City.*

Emery, Frederick E., Ph.D., Professor and Head of the *Department of Physiology and Pharmacology, University of Arkansas School of Medicine, Little Rock, Ark.*

Emmel, Victor M., A.B., Sc.M., Ph.D., Instructor in Anatomy, *The University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.*

Enders, Robert K., Ph.D., Associate Professor, *Edward Martin Biological Laboratory, Swarthmore College, Swarthmore, Pa.*

Engle, Earl Theron, A.B., A.M., Ph.D., Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 632 West 168th St., New York City.*

Esaki, Shiro, M.D., Ph.D., Member of Anatomical Institute, *Keio University, Sasuke 666, Kamakura, Japan.*

Essenberg, J. M., Ph.D., *The Chicago Medical College, 710 S. Wolcott Ave., Chicago, Ill.*

Essick, Charles Rhein, B.A., M.D., *700 Centre Ave., Reading, Pa.*

Etkin, William, Ph.D., Assistant Professor of Anatomy, *College of the City of New York, New York City.*

Ettinger, George Harold, B.A., M.D.C.M., F.R.S.C., Professor of Physiology, *Queen's University, Kingston, Ontario, Canada.*

Evans, F. Gaynor, B.A., M.A., Ph.D., Assistant Professor of Anatomy, *Wayne University, College of Medicine, Detroit 26, Mich.*

Evans, Herbert McLean, B.S., M.D., M.D. honoris causa, (Freiberg) Sc.D., (San Marcos) (Ex. Com. '19–'22; Pres. '30–'32), Hertzstein Professor of Biology, Professor of Anatomy and Director of the Institute of Experimental Biology, *University of California, Berkeley, Calif.*

EVANS, THOMAS HORACE, M.D., Research Professor of Anatomy, New York Medical College and Flower-Fifth Ave. Hospital; *350 South Main St., Freeport, N. Y.*

EVERETT, JOHN W., A.B., Ph.D., Assistant Professor of Anatomy, *Duke University School of Medicine, Durham, N. C.*

EVERETT, NEWTON B., B.S., M.S., Ph.D., Instructor in Anatomy, *University of Michigan, Ann Arbor, Mich.*

EVERSOLE, WILBURN J., A.B., M.Sc., Ph.D., Instructor of Biology, *The Rice Institute, Houston, Tex.*

FANKHAUSER, GERHARD, Ph.D., Associate Professor of Biology, *Princeton University, Princeton, N. J.*

FARIS, HERVEY S., A.B., A.M., Ph.D., M.D., Practicing Physician and Surgeon, *115 South Stone Ave., Tucson, Ariz.*

FARRIS, EDMOND J., Ph.D., Associate Member in Anatomy, Executive Director, *The Wistar Institute of Anatomy and Biology, Woodland Ave. and 36th St., Philadelphia 4, Pa.*

FIGGE, F. H. J., Ph.D., Associate Professor of Anatomy, *University of Maryland School of Medicine, Baltimore, Md.*

FINERTY, JOHN C., A.B., M.S., Ph.D., Instructor in Anatomy, *University of Michigan Medical School, Ann Arbor, Michigan.*

FLEXNER, LOUIS BARKHOUSE, S.B., M.D., Research Associate, *Department of Embryology, Carnegie Institution, Wolfe and Madison Sts., Baltimore, Md.*

FOGG, LLOYD C., B.S., M.S., Ph.D., Assistant Professor of Histology and Embryology, *Boston University Medical School, 80 E. Concord St., Boston, Mass.*

FOLEY, JAMES OWEN, M.S., Ph.D., Associate Professor of Anatomy, *Department of Anatomy, Medical College of Alabama, Birmingham 5, Ala.*

FORBES, THOMAS R., A.B., Ph.D., Instructor in Anatomy, *Yale University School of Medicine, 333 Cedar St., New Haven, Conn.*

FORMAN, JONATHAN, A.B., M.D., Professor Hist. Med., Lecturer on Diseases Due to Allergy, Ohio State University, *394 East Town St., Columbus, O.*

FOUST, H. L., D.V.M., Professor of Veterinary Anatomy, *Iowa State College, Ames, Ia.*

FOX, CLEMENT A., B.S., M.A., Ph.D., Assistant Professor of Anatomy, *Marquette University School of Medicine, Milwaukee 3, Wis.*

FRANCIS, C. C., M.D., Senior Instructor in Anatomy, *Western Reserve University School of Medicine, Cleveland, O.*

FRASER, DORIS ACORN, Ph.D., *333 South 22nd St., Philadelphia 3, Pa.*

FRAZER, JOHN ERNEST, D.Sc., F.R.C.S., Professor of Anatomy, University of London, *St. Mary's Hospital Medical School, London, W., England.*

FRENCH, H. E., M.S., M.D., Professor of Anatomy and Dean of the School of Medicine, *University of North Dakota, Grand Forks, N. Dak.*

FREUDENBERGER, CLAY B., Ph.D., M.D., Associate Dean and Professor of Anatomy, *University of Utah School of Medicine, Salt Lake City, Utah.*

FRIEDMAN, SYDNEY M., M.D., M.Sc., Assistant Professor of Anatomy, McGill University, *2101 University St., Montreal, Canada.*

GAMBLE, DEAN L., B.S., Ph.D., President, Ward's Natural Science Establishment, *P. O. Box 24, Beechwood Station, Rochester, N. Y.*

Garcia, Arturo, A.B., M.D., Professor of Anatomy and Head of Department, College of Medicine, University of the Philippines, *619 Colorado St., Manila, P. I.*

Gardner, Ernest, M.D., Assistant Professor of Anatomy, *School of Medicine, Wayne University, Detroit 26, Michigan.*

Gardner, William U., Ph.D., Professor of Anatomy and Chairman of the Department, *Yale University School of Medicine, 333 Cedar St., New Haven, Conn.*

Garrett, Frederic D., A.B., Ph.D., Instructor in Anatomy, *University of Nebraska, College of Medicine, 42nd and Dewey Ave., Omaha, Neb.*

Gatz, Arthur J., B.A., M.A., Ph.D., Assistant Professor of Anatomy, *Loyola University, School of Medicine, 706 So. Wolcott Ave., Chicago 12, Ill.*

Gaunt, Robert, Ph.D., Professor of Zoology and Chairman of the Department, *Lyman Hall, Syracuse University, Syracuse, N. Y.*

Geist, Frederick D., M.D., Associate Professor of Anatomy, *University of Wisconsin, Science Hall, Madison, Wis.*

George, Ruggles Kerr, B.A., M.B., D.P.H., Assistant Professor of Anatomy, *University of Toronto, Toronto 5, Canada.*

George, Wesley Critz, A.B., A.M., Ph.D., Professor of Histology and Embryology, and Head of the Department of Anatomy, *University of North Carolina Medical School, Chapel Hill, N. C.*

Gersh, Isidore, A.B., Ph.D., Associate Professor of Anatomy, *Johns Hopkins Medical School, Baltimore, Md.*

Gey, George O., B.S., M.D., Instructor in Surgery, Johns Hopkins Hospital and University, *Johns Hopkins Medical School, Baltimore, Md.*

Gillilan, Lois A., B.A., A.M., Ph.D., Instructor in Anatomy, *University of Pittsburgh Medical School, Department of Anatomy, Pittsburgh, Pa.*

Glickman, Irving, B.S., D.M.D., Assistant Professor of Oral Pathology, Tufts College Dental School, *416 Huntington Avenue, Boston, Mass.*

Globus, J. H., B.S., M.D., Associate Professor of Neuro-Anatomy, New York University, *960 Park Ave., New York City.*

Gluecksohn-Schoenheimer, Salome, Ph.D., Research Associate, *Department of Zoology, Columbia University, New York 27, N. Y.*

Goddard, James William (Elected as Goldner, Jacques Wilhelm), M.D., Pathelogist, *Long Island Hospital, Boston Harbor, Boston 13, Mass.*

Godwin, M. C., A.B., Ph.D., Associate Professor of Anatomy, Acting Head of the Dept. of Anatomy, *University of South Dakota, Medical School, Vermillion, S. Dak.*

Goldsmith, Eli D., M.A., M.S., Ph.D., Assistant Professor of Anatomy, *New York University, College of Dentistry, 477 First Avenue, New York 16, N. Y.*

Goldsmith, Joseph B., M.A., Ph.D., Associate Professor of Histology and Embryology, *Oklahoma University School of Medicine, Oklahoma City, Okla.*

Gomez, E. T., Ph.D., Assistant Physiologist, *Division of Nutrition and Physiology, Bureau of Dairy Industry, U. S. Department of Agriculture, Beltsville Research Center, Beltsville, Md.*

Goodrich, Hubert Baker, B.S., M.A., Ph.D., Professor of Biology, *Wesleyan University, Middletown, Conn.*

Gorbman, Aubrey, A.B., M.S., Ph.D., Res. Fellow in Anatomy, *Yale University School of Medicine, 333 Cedar St., New Haven 11, Conn.*

GORDON, ALBERT S., B.S., M.S., Ph.D., Assistant Professor of Biology, *Dept. Biology, Washington Square College of Arts and Science, New York University, N. Y.*

GOSS, CHARLES MAYO, A.B., M.D., Professor of Anatomy, Medical College of *Alabama, 620 So. 20th St., Birmingham 5, Ala.*

GOULD, HARLEY NATHAN, A.B., A.M., Ph.D., Professor of Biology, *Newcomb College, Tulane University, New Orleans, La.*

GRAFFLIN, ALLAN L., A.B., M.D., Assistant Professor of Anatomy, *Harvard Medical School, Boston, Mass.*

GRANT, J. C. BOILEAU, M.B., Ch.B., F.R.C.S. (Edin.), Professor of Anatomy, *University of Toronto, Toronto, Canada.*

GRANT, MADELEINE P., S.B., M.A., Ph.D., Member Teaching Staff in Zoology, *Sarah Lawrence College, Bronxville, N. Y.*

GRAVES, WILLIAM W., M.D., Professor and Director, Department of Neuropsychiatry, *St. Louis University School of Medicine, St. Louis, Mo.*

GRAY, DONALD J., B.S., M.S., Ph.D., Associate Professor of Anatomy, *Department of Anatomy, Stanford University, California.*

GRAY, MARY E., B.A., Ph.D., Instructor in Anatomy, *Vanderbilt University School of Medicine, Nashville, Tenn.*

GRAY, PETER, Ph.D., D.I.C., Associate Professor of Biology, *University of Pittsburgh, Pittsburgh, Pa.*

GREENE, CHARLES W., A.M., Ph.D., Professor Emeritus of Physiology and Pharmacology, University of Missouri, *814 Virginia Ave., Columbia, Mo.*

GREENE, EUNICE C., A.M., Research Investigator, *Syracuse University School of Medicine, Syracuse, N. Y.*

GREENE, WALTER FARRER, A.B., Ph.D., Associate Professor of Anatomy, *College of Medicine, Syracuse University, Syracuse, N. Y.*

GREENHILL, J. P., B.S., M.D., F.A.C.S., Professor and Vice Chairman, Department of Obstetrics and Gynecology, Loyola University Medical School; Professor of Gynecology, Cook County Graduate School of Medicine; Attending Gynecologist, Cook County Hospital, *Suite 2805–06 Pittsfield Building, 55 East Washington St., Chicago, Ill.*

GREEP, ROY O., M.S., Ph.D., Assistant Professor of Dental Science, Harvard School of Dental Medicine, Teaching Fellow, Dept. of Anatomy, *Harvard Medical School, 25 Shattuck St., Boston 15, Mass.*

GREGORY, WILLIAM KING, A.B., A.M., Ph.D., Sc.D., Da Costa Professor of Vertebrate Palaeontology, Columbia University; Curator Emeritus, Departments of Comparative Anatomy and Fishes, *American Museum of Natural History, New York City 24.*

GREULICH, WILLIAM W., Ph.B., M.A., Ph.D., Professor of Anatomy and Director of the Brush Foundation, Stanford University School of Medicine, *P. O. Box 1558, Stanford University, California.*

GROAT, RICHARD A., B.A., M.S., Ph.D., Assistant Professor of Neurology, *Institute of Neurology, Northwestern University Medical School, Chicago 11, Ill.*

GRODINSKY, MANUEL, B.Sc., M.D., Associate Professor of Anatomy and Surgery, University of Nebraska, College of Medicine, *681 N. 58th St., Omaha, Neb.*

GRUENWALD, PETER, M.D., Assistant in Pathology, *Kings County Hospital Division, Long Island College of Medicine, 451 Clarkson Ave., Brooklyn 3, N. Y.*

GUDERNATSCH, F., Ph.D., Visiting Professor, Graduate School, Department of Biology, *New York University, 100 Washington Square, New York 3, N. Y.*

GUILD, STACY R., A.M., Ph.D. (Ex. Com. '39–'46), Associate Professor of Otology and Director of Otological Research Laboratory, Johns Hopkins University, *Johns Hopkins Hospital, Baltimore, Md.*

GURDJIAN, ELISHA STEPHENS, A.B., M.S., M.D., Ph.D., F.A.C.S., Neuro-Surgeon, Grace Providence and Receiving Hospitals; Instructor in Surgery, Wayne University, *840 David Whitney Bldg., Detroit, Mich.*

GUTHRIE, MARY J., A.B., A.M., Ph.D., Professor of Zoology, *210 Lefevre Hall, University of Missouri, Columbia, Mo.*

GUTTMACHER, ALAN F., A.B., M.D., Associate Professor of Obstetrics, Johns Hopkins University, *1039 N. Calvert St., Baltimore, Md.*

GUYER, MICHAEL F., Ph.D., LL.D., Professor of Zoology, *University of Wisconsin, Madison, Wis.*

HALL, B. VINCENT, A.B., Ph.D., Associate Professor of Zoology, *University of Illinois, Urbana, Ill.*

HALL, BYRON E., Ph.D., M.D., *Mayo Clinic, Rochester, Minn.*

HALL, EDMUND K., D.Sc., Ph.D., Assistant Professor of Anatomy, *University of Louisville Medical School, Louisville, Ky.*

HALL, E. RAYMOND, A.B., M.A., Ph.D., Professor of Zoology, Chairman Department of Zoology, Director Museum of Natural History, *University of Kansas, Lawrence, Kansas.*

HALPERT, BÉLA, M.D., Director of Laboratories of the University Hospitals and Professor of Clinical Pathology, *University of Oklahoma School of Medicine, 800 East 13th St., Oklahoma City 4, Okla.*

HAM, ARTHUR W., M.B., Associate Professor of Anatomy, *University of Toronto, Toronto, Canada.*

HAMBLEN, E. C., M.D., Associate Professor of Obstetrics and Gynecology, *Duke University School of Medicine, Durham, N. C.*

HAMBURGER, VIKTOR, Ph.D., Professor of Zoology, *Washington University, St. Louis, Mo.*

HAMILTON, JAMES B., Ph.D., Professor of Anatomy, Long Island College of Medicine, *350 Henry St., Brooklyn 2, New York.*

HAMLETT, GEORGE W. D., A.B., A.M., Ph.D., Research Agent, *U. S. Biological Survey, Department of Embryology, Carnegie Institution of Washington, Baltimore, Md.*

HAMMOND, WARNER S., Ph.D., Assistant Professor of Anatomy, *Cornell University Medical College, New York City 21.*

HANAN, ERNEST B., A.B., A.M., M.D., Chief Pathologist of Burge Hospital School of Nursing, *600 Medical Arts Building, Springfield, Mo.*

HARD, WALTER LEON, A.M., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, Medical College of the State of South Carolina, Charleston 16, S. C.*

HARE, W. KENDRICK, A.B., M.S., Ph.D., Associate Professor of Physiology, *State University of Iowa, Iowa City, Ia.*

HARGITT, GEORGE T., A.B., M.S., Ph.D., Sc.D., Professor of Zoology, *Biology Bldg., Duke University, Durham, N. C.*

HARMAN, PINCKNEY J., B.S., M.S., Ph.D., Instructor in Anatomy, *Georgetown University School of Medicine, 3900 Reservoir Rd., N. W., Washington, D. C.*

HARRIS, HENRY A., M.A., M.D., B.S., M.R.C.P., D.Sc., Professor of Anatomy, *The Anatomy School, The University, Cambridge, England.*

HARRISON, FRANK, B.S., M.S., Ph.D., Associate Professor of Anatomy, *University of Tennessee, Memphis, Tenn.*

HARRISON, ROSS GRANVILLE, Ph.D., M.D., Sc.D., LL.D., (Pres. '12–'14), Chairman National Research Council; Sterling Professor of Biology and Director of Osborn Zoological Laboratory Emeritus, *Yale University, New Haven, Conn.*

HARTMAN, CARL G., M.A., Ph.D. (Ex. Com. '29–'33) (First Vice-Pres. '40–'42), Professor of Zoology, Head of Department of Zoology and Physiology, Chairman Division of Biological Sciences, *University of Illinois, Urbana, Ill.*

HARTMANN, J. FRANCIS, A.B., Ph.D., Instructor in Anatomy, *Institute of Anatomy, University of Minnesota, Minneapolis 14, Minn.*

HARVEY, BASIL COLEMAN HYATT, A.B., M.D., Professor of Anatomy Emeritus, *The University of Chicago, Chicago, Ill.*

HASHIMOTO, EDWARD I., A.B., M.D., M.S., Associate Professor of Anatomy, *University of Utah Medical School, Salt Lake City, Utah.*

HATERIUS, HANS OLIVER, A.B., M.S., Ph.D., Professor of Physiology, *Boston University School of Medicine, 80 E. Concord, Boston 18, Mass.*

HAUSMAN, LOUIS, A.B., M.D., Associate Professor of Clinical Neurology, Cornell University Medical College; Associate Attending Physician, New York Hospital, *140 E. 54th St., New York 22, N. Y.*

HEGRE, ERLING S., B.A., M.S., Ph.D., Associate Professor of Anatomy, *Department of Anatomy, Medical College of Virginia, Richmond 19, Virginia.*

HEINBECKER, PETER, M.D., Associate Professor of Surgery, *Washington University Medical School, St. Louis, Mo.*

HELDT, THOMAS J., A.B., A.M., M.D., Physician in Charge of Division of Neuropsychiatry, *Henry Ford Hospital, Detroit, Mich.*

HELFF, O. M., B.S., M.S., Ph.D., Associate Professor of Biology, *New York University, University Heights, New York City.*

HELLBAUM, ARTHUR A., Ph.D., M.D., Professor of Pharmacology, *University of Oklahoma, School of Medicine, Oklahoma City, Okla.*

HERRICK, CHARLES JUDSON, Ph.D., (Ex. Com. '13–'17), Emeritus Professor of Neurology, The University of Chicago, *236 Morningside Drive, Grand Rapids 6, Mich.*

HERTIG, ARTHUR TREMAIN, B.S., M.D., Assistant Professor of Pathology and Assistant Professor of Obstetrics, Harvard Medical School; Pathologist and Visiting Obstetrician to Outpatients, Boston Lying-in Hospital; Pathologist, Free Hospital for Women, Brookline, Mass; *221 Longwood Ave., Boston 15, Mass.*

HERTZLER, ARTHUR E., B.S., A.M., Ph.D., M.D., LL.D., D.Sc., F.A.C.S., Professor of Surgery, University of Kansas, *Halstead, Kan.*

HETHERINGTON, ALBERT W., B.S., Ph.D., Altitude Physiologist, *A. A. F. School of Aviation Medicine, Randolph Field, Texas.*

HETHERINGTON, DUNCAN CHARTERIS, A.B., M.A., Ph.D., M.D., Associate Professor of Anatomy, *Duke University Medical School, Durham, N. C.*

Heuser, Chester H., A.M., Ph.D., Research Associate, Department of Embryology, Carnegie Institution, *Johns Hopkins Medical School, Baltimore, Md.*

Higgins, George M., A.M., Ph.D., Associate Professor of Experimental Biology, *University of Minnesota, Mayo Foundation, Rochester, Minn.*

Hill, James Peter, D.Sc., F.R.S., Professor Emeritus of Embryology, Department of Anatomy, University College, London, W. C. 1, *Kanimbla Dollis Ave., Finchley, London, W. 3, England.*

Hill, John E., A.B., Ph.D., Assistant Curator of Mammals, *American Museum of Natural History, 79th St. and Central Park West, New York City.*

Hill, Robert T., Ph.D., Associate Professor of Anatomy, *Indiana University, Medical School, Bloomington, Ind.*

Hilton, William A., B.S., Ph.D., Professor of Zoology, Pomona College, Director Laguna Marine Laboratory, *1263 Dartmouth Ave., Claremont, Calif.*

Hines, Marion, A.B., Ph.D., Sc.D., (2nd Vice-Pres. '46–) Associate Professor of Anatomy, *Johns Hopkins Medical School, Baltimore 5, Md.*

Hinsey, Joseph C., M.S., Ph.D., (Ex. Com. '40–), Dean and Professor of Anatomy, *Cornell University Medical College, 1300 York Ave., New York City 21.*

Hirsch, G. Christian, Ph.D., Lector, Head of Department of Experimental Histology, *Zoological Laboratory. The University, Utrecht, Janskerkhof 3, Holland.*

Hisaw, Frederick L., A.B., A.M., Ph.D., LL.D., Professor of Zoology, Chairman of the Department of Biology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Hoadley, Leigh, A.B., Ph.D., Professor of Zoology, *Harvard University, Cambridge, Mass.*

Hoerr, Normand L., A.B., Ph.D., M.D., (Sec.-Treas. '46–) Professor of Anatomy, *School of Medicine, Western Reserve University, 2109 Adelbert Rd., Cleveland 16, O.*

Hogg, Ira D., B.A., M.A., Ph.D., Assistant Professor of Anatomy, Department of Anatomy, University of Pittsburgh School of Medicine, *617 Hampton Ave., Wilkinsburg, Pa.*

Hogue, Mary J., Ph.D., Associate in Anatomy, *University of Pennsylvania Medical School, Philadelphia 4, Pa.*

Hollinshead, W. Henry, A.B., M.S., Ph.D., Associate Professor of Anatomy, *Duke University School of Medicine, Durham, N. C.*

Holt, Caroline M., A.B., A.M., Ph.D., Professor of Biology Emerita, *Simmons College, 38 Ridge Ave., Newton Centre, Mass.*

Holyoke, Edward A., M.D., Ph.D., Associate Professor of Anatomy, *University of Nebraska, College of Medicine, 42nd and Dewey Ave., Omaha, Neb.*

Hooker, Charles W., Ph.D., Assistant Professor of Anatomy, *Yale University School of Medicine, New Haven, Conn.*

Hooker, Davenport, Ph.D., Sc.D. (Ex. Com., '22–'25) (Second Vice-Pres. '40–'42), Professor of Anatomy and Head of the Department, *School of Medicine, University of Pittsburgh, Pittsburgh, Pa.*

Hopkins, Grant Sherman, Sc.D., D.V.M., Emeritus Professor of Comparative Veterinary Anatomy, *Cornell University, Ithaca, N. Y.*

Hoskins, Margaret Morris, Ph.D., Associate Professor of Microscopic Anatomy, *New York University Dental College, 477 First Ave., New York City.*

House, Earl Lawrence, B.S., M.A., Ph.D., Assistant Professor of Anatomy, Essex College of Medicine and Surgery, *105 Broad Street, Newark 4, N. J.*

Howe, Howard A., M.D., Associate Professor of Epidemiology, *Johns Hopkins University School of Hygiene, Baltimore, Md.*

Howland, Ruth B., Ph.D., Associate Professor of Biology, Washington Square College, *New York University, Washington Square East, New York City.*

Hoy, Wm. E., A.B., Ph.D., Professor of Biology and Head of the Department of Biology, *University of South Carolina, Columbia, S. C.*

Huber, John Franklin, A.M., M.D., Ph.D., Professor and Head of the Department of Anatomy, *School of Medicine, Temple University, Broad St. at Ontario, Philadelphia 40, Pa.*

Humm, Frances Dorris, Ph.D., Research Assistant in Embryology, *Yale University, BG. 22 Sterling Hall of Medicine, New Haven, Conn.*

Hummel, Katharine P., Ph.D., Research Associate, *Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.*

Humphrey, Rufus R., A.M., Ph.D., Professor of Anatomy, School of Medicine, University of Buffalo, *24 High St., Buffalo, N. Y.*

Humphrey, Tryphena, M.D., Ph.D., Assistant Professor of Anatomy, *University of Pittsburgh School of Medicine, Pittsburgh, Pa.*

Hunt, Thomas E., A.B., Ph.D., Associate Professor of Histology and Embryology, *Medical College of Alabama, Birmingham 5, Ala.*

Hunter, Oscar B., A.B., A.M., M.D., Professional Lecturer, Georgetown University, *1835 Eye St., N. W., Washington, D. C.*

Hutchinson, Cranford, Ph.D., Associate in Anatomy, *Daniel Baugh Institute of Anatomy, Jefferson Medical College, Philadelphia 7, Pa.*

Ingalls, N. William, B.S., M.D., Associate Professor of Anatomy, School of Medicine, Western Reserve University, *3526 Edison Road, Cleveland Heights, O.*

Ingersoll, Everett H., B.S., M.S., Ph.D., Associate Professor of Anatomy, *Medical College of Virginia, Richmond, Va.*

Ingram, W. R., M.S., Ph.D., Professor and Head, Department of Anatomy, *State University of Iowa, College of Medicine, Iowa City, Ia.*

Jackson, Clarence M., M.S., M.D., LL.D., (Ex. Com. '10–'14, '23–'28, Vice-Pres. '16–'17, Pres. '22–'23), Professor Emeritus of Anatomy, *Institute of Anatomy, University of Minnesota, Minneapolis, Minn.*

Jackson, Joseph L., M.A., M.B., Professor of Anatomy, *University of Saskatchewan, Saskatoon, Canada.*

Jailer, Joseph William, B.S., M.S., Ph.D., M.D., Captain, M. C. Chief, Department of Chemistry, *Army Medical School, Army Medical Center, Washington 12, D. C.*

Janes, Ralph G., A.B., B.S., M.S., Ph.D., Assistant Professor of Anatomy, *Dept. of Anatomy, State University of Iowa, College of Medicine, Iowa City, Iowa.*

Jeffers, Katharine R., Ph.D., Assistant Professor of Zoology, *Duke University, Durham, N. C.*

Jenkins, George B., M.D., Emeritus Professor of Anatomy and Director of Anatomy Department, *George Washington University Medical School, Washington, D. C.*

Job, Thesle T., A.B., M.S., Ph.D., Professor of Anatomy, *Loyola University School of Medicine, 706 South Wolcott Ave., Chicago, Ill.*

Johnson, Myra L., A.B., A.M., Ph.D., *161 High Street, Wadsworth, Ohio.*

Johnson, Percy L., B.S., M.S., Ph.D., Assistant Professor of Anatomy, *New York University Dental College, 477 First Avenue, New York 16, N. Y.*

Johnson, Sydney E., Ph.D., M.D., F.A.C.P., Professor of Gross Anatomy, School of Medicine, University of Louisville, *101 West Chestnut St., Louisville, Ky.*

Johnston, Thomas Baillie, C.B.E., M.D., Professor of Anatomy, *Guy's Hospital Medical School, London University, London, S. E. 1, England.*

Johnstone, Paul N., A.B., M.D., Instructor in Surgery, University of Kansas Medical School, *210 Argyle Bldg., Kansas City. Mo.*

Jones, David S., Ph.D., Assistant Professor of Anatomy, *Loyola University Medical School, 706 S. Wolcott St., Chicago, Ill.*

Jones, Oliver P., A.B., Ph.D., Professor and Head of the Department of Anatomy, *University of Buffalo, School of Medicine, 24 High St., Buffalo, N. Y.*

Jones, Russell, Ph.D., Assistant Professor of Anatomy, *Indiana University School of Medicine, Bloomington, Ind.*

Jordan, Harvey Ernest, A.M., Ph.D., Sc.D., (Ex. Com. '18–'21) (First Vice-Pres. '36–'38), Dean of the Department of Medicine, Professor of Anatomy and Director of the Anatomical Laboratories, *Pavilion VIII, East Lawn, University of Virginia, Charlottesville, Va.*

Joy, Edward A., A.B., A.M., Ph.D., Associate Professor of Anatomy, *Tufts College Medical School, 416 Huntington Ave., Boston, Mass.*

Juhn, Mary, Ph.D., Research Professor, Department of Poultry Husbandry, *University of Maryland, College Park, Maryland.*

Kaan, Helen Warton, Ph.D., Associate Professor of Zoology, *Wellesley College, Wellesley 81, Mass.*

Kabat, Herman, Ph.D., M.D., Consultant in Neurology, Crippled Children's Program, *Health Department, District of Columbia, Washington, D. C.*

Kammeraad, Adrian, A.B., Ph.D., Director of Research, Von Patten Pharmaceutical Co., *500 N. Dearborn St., Chicago 10, Ill.*

Kampmeier, Otto Frederic, A.B., Ph.D., M.D., Professor of Anatomy and Head of Department, *College of Medicine, University of Illinois, 1853 W. Polk St., Chicago, Ill.*

Kaplan, Emanuel B., B.S., M.D., Instructor in Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32, N. Y.*

Kappers, Cornelius Ubbo Ariëns, M.D., Sc.D., T.D., Director of the Central T.L.L. Institute for Brain Research of Holland; *Professor of Comparative Neurology, University of Amsterdam, Mauritskade 61, Amsterdam, Holland.*

Kaylor, Cornelius T., A.B., M.S., Ph.D., Assistant Professor of Anatomy, *College of Medicine, Syracuse University, Syracuse, N. Y.*

Keith, Sir Arthur, M.D., LL.D., F.R.C.S., D.Sc., F.R.S., Master of *Buckston Browne Research Farm, Downe, Kent, England.*

Kellogg, Remington, Ph.D., Curator, Division of Mammals, *U. S. National Museum, Washington, D. C.*

KELLY, G. LOMBARD, A.B., B.S., M.D., Dean and Professor of Anatomy, *University of Georgia, School of Medicine, Augusta, Ga.*

KERNAN, JOHN D., JR., A.B., M.D., Professor of Oto-Laryngology, Columbia College of Physicians and Surgeons, *103 East 78th St., New York City.*

KEY, J. ALBERT, B.S., M.D., Clinical Professor of Orthopedic Surgery, *Washington University, St. Louis, Mo.*

KILLE, FRANK R., Ph.D., Professor of Zoology and Dean of Men, *Carleton College, Northfield, Minn.*

KIMMEL, DONALD L., Ph.D., Associate Professor of Anatomy, *School of Medicine, Temple University, Broad St. at Ontario, Philadelphia, Pa.*

KINDRED, JAMES ERNEST, A.B., A.M., Ph.D., Associate Professor of Anatomy, *University of Virginia Medical School, Box 1873, University Station, Charlottesville, Va.*

KING, HELEN DEAN, A.M., Ph.D., Member of The Wistar Institute (Experimental Zoology), *The Wistar Institute of Anatomy, 36th St. and Woodland Ave., Philadelphia 4, Pa.*

KING, JESSIE L., Ph.D., Professor of Physiology, *Goucher College, Baltimore, Md.*

KINGERY, HUGH MCMILLAN, A.B., A.M., Ph.D., Associate Professor of Anatomy, *School of Medicine, University of Colorado, Denver, Colo.*

KINGSBURY, BENJAMIN F., A.B., Ph.D., M.D., D.Sc., (Ex. Com. '22–'25, Vice-Pres. '32–'34), Professor Emeritus of Histology and Embryology, Cornell University, Ithaca, N. Y., *211 Glenburnie St., Chapel Hill, N. C.*

KINGSLEY, D. M., M.S., Ph.D., M.D., Assistant Visiting Surgeon to Charity Hospital, *15 Fontainebleau Drive, New Orleans, La.*

KIRGIS, HOMER D., M.D., Ph.D., Instructor in Anatomy, *Tulane University School of Medicine, New Orleans 15, Louisiana.*

KIRKHAM, WILLIAM BARRI, Ph.D., Consulting Director, Museum of Natural History, *220 State St., Springfield 5, Mass.*

KIRKMAN, HADLEY, A.B., S.M., Ph.D., Associate Professor of Anatomy, *Stanford University, Calif.*

KIRSCHBAUM, ARTHUR, Ph.D., M.D., Associate Professor of Anatomy, *Institute of Anatomy, University of Minnesota, Minneapolis, Minn.*

KNISELY, MELVIN H., Ph.D., Associate Professor of Anatomy, *The University of Chicago, Chicago, Ill.*

KNOUFF, R. A., B.A., M.A., Ph.D., Professor of Anatomy, *Department of Anatomy, Ohio State University, Columbus, O.*

KOCH, JOHN C., B.S., M.D., *1053 Iroquois Ave., Detroit 14, Mich.*

KOFOID, CHARLES ATWOOD, A.B., Ph.D., Sc.D., LL.D., Professor of Zoology Emeritus, *Room 5090, Life Sciences Bldg., University of California, Berkeley, Calif.*

KOLOUCH, FRED, JR., A.B., M.A., M.B., M.D., Fellow of the National Science Foundation, *University of Minnesota Medical School, Minneapolis 14, Minn.*

KONEFF, ALEXEI ALEXANDER, M.D., Assistant Professor of Anatomy and Lecturer in Histological Technique, *University of California Medical School, Life Sciences Building, Berkeley 4, Calif.*

KORNHAUSER, S. I., M.A., Ph.D., (Ex. Com. '42–), Professor of Anatomy and Executive Secretary, *University of Louisville, School of Medicine, 101 W. Chestnut St., Louisville, Ky.*

KRAFKA, JOSEPH, JR., Ph.D., M.D., Professor of Microscopic Anatomy, *University of Georgia School of Medicine, Augusta, Ga.*

KRAMER, THEODORE C., B.S., M.A., Ph.D., Instructor in Anatomy, *University of Michigan, Ann Arbor, Mich.*

KREHBIEL, ROBERT H., M.S., Ph.D., Associate Professor of Anatomy, *University of Illinois, College of Medicine, 1853 W. Polk St., Chicago 12, Ill.*

KRIEG, WENDELL J. S., B.S., M.S., Ph.D., Associate Professor of Neurology, *Institute of Neurology, Northwestern University Medical School, 303 E. Chicago Ave., Chicago, Ill.*

KROGMAN, WILTON M., Ph.D., Associate Professor of Anatomy and Physical Anthropology, *Department of Anatomy, The University of Chicago, Chicago 11, Ill.*

KROPP, BENJAMIN, Ph.D., Assistant Professor of Embryology, *Queen's University Medical School, Kingston, Ontario, Canada.*

KUHLENBECK, HARTWIG, M.D., Ph.D., Professor of Anatomy, *Woman's Medical College of Pennsylvania, Philadelphia, Pa.*

KUNKEL, BEVERLY WAUGH, Ph.B., Ph.D., Professor of Biology, *Lafayette College, Easton, Pa.*

KUNTZ, ALBERT, Ph.D., M.D. (Second Vice-Pres. '38–'39; First Vice-Pres. '39–'40), Professor of Micro-anatomy, *St. Louis University Medical School, 1402 South Grand Ave., St. Louis, Mo.*

KUTCHIN, MRS. HARRIET LEHMANN, A.M., *"The Maplewood," Green Lake, Wis.*

LACHMAN, ERNEST, M.D., Professor of Anatomy, Chairman of the Department, *University of Oklahoma School of Medicine, Oklahoma City, Okla.*

LAMBERT, AVERY ELDORUS, B.S., Ph.D., Professor of Histology, State University of Iowa Medical School, *Room 360, Medical Laboratories Bldg., Iowa City, Ia.*

LANGWORTHY, ORTHELLO R., A.B., A.M., M.D., Associate Professor of Psychiatry, Johns Hopkins University, *Johns Hopkins Hospital, Baltimore, Md.*

LANIER, RAYMOND R., Ph.D., M.D., Resident, Division of Roentgenology, *The University of Chicago Clinics, Chicago 37, Ill.*

LARSELL, OLOF, A.M., Ph.D., (Ex. Com. '38–'42), Professor of Anatomy, Dean of Graduate Division, Oregon State System of Higher Education, *University of Oregon Medical School, Portland, Ore.*

LASSEK, ARTHUR M., M.S., Ph.D., M.D., Professor of Anatomy, *Medical College of the State of South Carolina, Charleston, S. C.*

LATIMER, HOMER B., A.M., Ph.D., Professor of Anatomy, *Medical School, University of Kansas, Lawrence, Kan.*

LATTA, JOHN S., A.B., Ph.D., Professor of Anatomy, University of Nebraska, College of Medicine, *Forty-second St. and Dewey Ave., Omaha, Neb.*

LAUER, EDWARD W., B.S., M.A., Ph.D., Instructor in Anatomy, *Department of Anatomy, E. Medical Bldg., University of Michigan, Ann Arbor, Michigan.*

LAWLESS, JOHN J., M.A., Ph.D., Director, University Health Service, *West Virginia University, Morgantown, W. Va.*

LAZAROW, ARNOLD, M.D., Ph.D., Senior Instructor in Anatomy, *School of Medicine, Western Reserve University, 2109 Adelbert Rd., Cleveland 61, O.*

LEATHEM, JAMES H., B.S., M.A., Ph.D., Assistant Professor of Zoology, *Rutgers University, New Brunswick, N. J.*

Leblond, Charles Philippe, M.D., Ph.D., Associate Professor of Microscopic Anatomy, *Department of Anatomy, McGill University, Montreal, Canada.*

Lee, Ferdinand Christian, A.B., M.D., Associate Professor of Surgery, *Johns Hopkins Hospital, Baltimore, Md.*

Leonard, Samuel L., Ph.D., Associate Professor of Zoology, *Cornell University, Ithaca, N. Y.*

Levenstein, Irving M., Ph.D., Endocrinologist, *Leberco Laboratories, Bloomfield, N. J.*

Levi, Giuseppe, M.D., Professor of Anatomy, *Institute Anatomica, the University of Turin, Corte D'Areglia 52, Torina, Italy.*

Lewis, Frederic T., A.M., M.D., (Ex. Com. '09–'13, Vice-Pres. '14–'16; Pres. '36–'38), Professor of Comparative Anatomy, Emeritus, *Harvard Medical School, Boston, Mass.*

Lewis, Margaret Reed, *The Wistar Institute, 36th St. and Woodland Ave., Philadelphia 4, Pa.*

Lewis, Warren Harmon, B.S., M.D., (Ex. Com. '09–'11, '14–'17, Pres. '34–'36), *The Wistar Institute, 36th St. and Woodland Ave., Philadelphia 4, Pa.*

Liedke, Kathe B., A.B., M.Sc., Ph.D., Associate, *New York Medical College, Flower and Fifth Ave. Hospitals, Fifth Ave. and 105th St., New York City.*

Lillie, Frank Rattray, Ph.D., Sc.D., LL.D., Andrew MacLeish Distinguished Service Professor Embryology Emeritus, The University of Chicago, *5801 Kenwood Ave., Chicago, Ill.*

Limson, Marciano, M.D., Associate Professor of Anatomy, *College of Medicine, University of the Philippines, Manila, P. I.*

Linell, Eric Ambrose, Ch.B., M.D., Professor of Neuropathology, *Banting Institute, University of Toronto, Canada.*

Lipshutz, Benjamin, M.D., Assistant Professor in Neuro-anatomy, Jefferson Medical College, *1007 Spruce St., Philadelphia, Pa.*

Little, Clarence Cook, B.A., M.S., Sc.D., LL.D., Litt.D., Director, *Jackson Memorial Laboratory, Bar Harbor, Me.*

Lloyd, Ruth Smith, A.B., M.S., Ph.D., Instructor in Anatomy, *Howard University, Washington, D. C.*

Lobo, Bruno Alipio, B.A., M.D., ''Docente Livre'' Professor of Histology and Embryology, *Faculdade Nacional de Medicine da Universidade do Brasil, Avenida Pasteur, Rio de Janeiro, Brasil.*

Long, Margaret Eleanor, Ph.D., Instructor in Anatomy, *Medical College of Alabama, 620 South 20th Street, Birmingham 5, Alabama.*

Looper, James Burdine, Ph.D., M.D., Professor of Anatomy, *University of Mississippi, Oxford, Miss.*

Loosli, Clayton G., M.D., Ph.D., Associate Professor of Medicine, Dept. of Medicine, The University of Chicago, *950 E. 59th St., Chicago 37, Ill.*

Lord, Frederic P., A.B., M.D., Professor of Anatomy, *Dartmouth Medical School, 39 College St., Hanover, N. H.*

Lorente de Nó, Raphael, M.D., Member, *Rockefeller Institute for Medical Research, 66th St. and York Ave., New York City 21.*

Loth, Edward, M.D., Ph.D., Professor of Anatomy and Director of Anatomical Institute, University of Warsaw, *5 Chalubinski Str., Warsaw, Poland.* Deceased.

LOW, FRANK NORMAN, A.B., Ph.D., Associate Professor of Anatomy, *West Virginia University, School of Medicine, Morgantown, W. Va.*

LOWERY, LAWSON GENTRY, A.M., M.D., Psychiatrist, Brooklyn Hebrew Orphan Asylum and Travelers Aid Society, *25 West 54th St., New York City.*

LUCAS, ALFRED M., Ph.D., Cytopathologist, *U. S. Regional Poultry Research Laboratory, E. Mt. Hope Rd., East Lansing, Mich.*

LUCAS, MIRIAM SCOTT, Ph.D., Research, *521 Elizabeth St., East Lansing, Mich.*

LYONS, WILLIAM R., B.A., M.A., Ph.D., Associate Professor of Anatomy, *University of California, Berkeley, Calif.*

MACCALLUM, DANIEL BARTLETT, S.B., Ph.D., M.D., Professor of Anatomy, *University of Southern California Medical School, Los Angeles, Calif.*

MACCARDLE, ROSS C., B.S., Ph.D., Instructor in Anatomy and Research Associate, *Washington University School of Medicine, St. Louis, Mo.*

MACEWEN, EWEN, B.S., M.S., M.D., Professor of Anatomy, Director of the Anatomical Laboratories, Dean, College of Medicine, *University of Iowa, Iowa City, Ia.*

MACFALL, CLAUDE M., Ph.D., Professor of Anatomy, *George Washington Medical School, 1335 H Street, N. W., Washington, D. C.*

MACFARLAND, FRANK MACE, A.M., Ph.D., Emeritus Professor of Histology, Leland Stanford Junior University; President California Academy of Sciences, San Francisco; *775 Santa Ynez St., Stanford University, Calif.*

MACFARLAND, WILLIAM ELLERY, B.A., Ph.D., Editor, *W. B. Saunders and Company, Seventh and Locust Streets, Philadelphia, Pa.*

MACKLIN, C. C., M.B., M.D., M.A., Ph.D., F.R.S.C. (Ex. Com. '28–'32), Professor of Histology and Embryology, *University of Western Ontario Medical School, London, Ontario, Canada.*

MACKLIN, MADGE THURLOW, A.B., M.D., LL.D., Senior Research Fellow, National Research Council, *Ohio State University, Columbus, Ohio.*

MACLEAN, BERNICE L., A.B., Ph.D., Assistant Professor of Zoology, Department of Biological Sciences, *Hunter College, 695 Park Ave., New York City.*

MACLEOD, JOHN, A.B., M.S., Ph.D., Research Associate in Anatomy, *Cornell University Medical College, 1300 York Ave., New York City.*

MAGATH, THOMAS BYRD, M.S., Ph.D., M.D., Consulting Physician, Clinical Laboratories, Mayo Clinic and Professor of Pathology and Parasitology, University of Minnesota, *Mayo Clinic, Rochester, Minn.*

MAGOUN, HORACE W., Ph.D., Professor of Microscopic Anatomy, *Northwestern University Medical School, 303 E. Chicago Ave., Chicago, Ill.*

MAGRUDER, SAMUEL R., B.S., M.A., Ph.D., Assistant Professor of Anatomy, *Tufts College Medical School, 416 Huntington Ave., Boston, Mass.*

MAHON, ELEANOR CONWAY, Ph.D., *Iron River, Mich.*

MAINLAND, DONALD, M.B., Ch.B., D.Sc., F.R.S.E., F.R.S.C., Professor of Anatomy and Head of the Department, *Dalhousie University, Halifax, Nova Scotia, Canada.*

MALONE, EDWARD F., A.B., M.D., (Ex. Com. '31–'35), Francis Brunning Professor of Anatomy, *153 Cortland Ave., Winter Park, Florida.*

MANTER, JOHN T., A.B., Ph.D., Assistant Professor of Microscopic Anatomy, *University of Georgia, School of Medicine, Augusta, Ga.*

MARBURG, OTTO, M.D., Clinical Professor of Neurology, Columbia University College of Physicians and Surgeons, *225 Central Park West, New York, N. Y.*

Mark, Edward Laurens, Ph.D., LL.D., Hersey Professor of Anatomy (Emeritus), Harvard University and Trustee of the Bermuda Biological Station for Research, Inc., *109 Irving St., Cambridge, Mass.*

Markee, J. E., B.S., Ph.D., Professor of Anatomy, Head of Anatomy Department, *Duke University School of Medicine, Durham, N. C.*

Marshall, Clyde, M.D., *25 Beech St., Halifax, Nova Scotia, Canada.*

Martin, Cecil P., M.A., M.B., D.Sc., Professor of Anatomy, *McGill University, Montreal, Canada.*

Martin, John, B.S., M.S., M.B., M.D., Associate in Surgery, *Northwestern University Medical School, Chicago, Ill.*

Marvin, Horace Newell, B.A., M.A., Ph.D., Assistant Professor of Anatomy, *University of Arkansas, School of Medicine, Little Rock, Arkansas.*

Mason, Karl E., Ph.D., Professor of Anatomy, *The University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.*

Masson, Georges M. C., D.V.M., Ph.D., Professeur Agrégé, Institut de Médecine et de Chirugie experimentales, *Université de Montréal, Montréal, Canada.*

Mastin, E. Vernon, M.D., M.S. (in Surgery), Assistant Professor of Surgery, St. Louis University Medical School, *3720 Washington Ave., St. Louis, Mo.*

Matas, Rudolph, M.D., LL.D., Sc.D., F.R.C.S. Eng. (hon.), (Charter member), Professor of Surgery (1894–1927), Emeritus (1927–), and Demonstrator of Anatomy (1885–1894), Tulane University, *2255 St. Charles Ave., New Orleans 13, La.*

Matthews, Samuel A., M.A., Ph.D., Professor of Biology, *Williams College, Williamstown, Mass.*

Mavor, James Watt, B.A., M.A., Ph.D., Professor of Biology, *24 Edward Street, Belmont 78, Mass.*

Mayer, Edmund, M.D., Pathologist, Chemotherapy Division, Stamford Research Laboratories, *American Cyanamid Company, 1937 W. Main Street, Stamford, Conn.*

McClendon, Jesse F., Ph.D., Research Professor of Physiology, Hahnemann Medical College, *Route 1, Trooper Rd., Norristown, Pa.*

McClure, Charles Freeman Williams, A.M., Sc.D. (Vice-Pres. '10–'11, Ex. Com. '12–'16, Pres. '20–'21), Professor Emeritus of Comparative Anatomy, *Princeton University, 1 Battle Road, Princeton, N. J.*

McCotter, Rollo E., M.D., Professor of Anatomy, Medical School, University of Michigan, *Lakewood R. R. 1, Ann Arbor, Mich.*

McCrady, Edward, Jr., A.B., M.S., Ph.D., Professor of Biology, *University of the South, Sewanee, Tenn.*

McCulloch, Warren S., A.B., A.M., M.D., Associate Professor of Psychiatry, College of Medicine, University of Illinois, *912 South Wood St., Chicago, Ill.*

McCullough, Albert William, A.B., B.Sc., M.A., Ph.D., Associate Professor of Anatomy, *University of Arkansas School of Medicine, Little Rock, Arkansas.*

McGill, Caroline, A.B., A.M., Ph.D., M.D., Physician, *58 W. Quartz, Butte, Mont.*

McKenzie, Fred F., B.S.A., A.M., Ph.D., D.Agr., D.V.S., Professor of Animal Husbandry, *Oregon State College, Corvallis, Ore.*

McKinney, Roscoe L., Ph.D., Professor of Anatomy, *Howard University, Washington, D. C.*

Mead, Harold T., Ph.D., Professor of Biology, *Tennessee College for Women, Murfreesboro, Tenn.*

Meader, Ralph G., Ph.D., Lecturer, Associate Professor in Anatomy, Assistant Director Board of Scientific Advisors, The Jane Coffin Childs Memorial Fund for Medical Research, *Yale University School of Medicine, 333 Cedar St., New Haven, Conn.*

Meaker, Samuel R., A.B., A.M., M.D., M.R.C.S. (Eng.), F.A.C.S., F.C.O.G., Professor of Gynaecology, Boston University School of Medicine, *475 Commonwealth Ave., Boston, Mass.*

Mendelsohn, William, M.S., M.D., Clinical Instructor in Surgery, Yale Medical School, *442 Temple St., New Haven, Conn.*

Menkin, Valy, B.S., M.A., M.D., Associate Professor of Experimental Pathology, Department of Surgical Research, *Temple University Medical School, Broad Street at Ontario, Philadelphia 40, Pa.*

Merriman, Daniel, B.S., M.S., Ph.D., Assistant Professor of Biology and Director, Bingham Oceanographic Laboratory, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

Mettler, Fred. A., A.M., M.D., Ph.D., Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University, *630 West 168th St., New York City.*

Meyer, Adolf, M.D., LL.D., Professor Emeritus in Psychiatry, Johns Hopkins Hospital, *4305 Rugby Rd., Baltimore 10, Md.*

Meyer, Arthur W., S.B., M.D., (Ex. Com. '12–'16, Vice-Pres. '24–'25), Professor of Anatomy Emeritus, Leland Stanford Junior University, *121 Waverly St., Palo Alto, Calif.*

Meyer, Roland K., B.A., M.A., Ph.D., Professor of Zoology, *Department of Zoology, University of Wisconsin, Madison, Wis.*

Michels, Nicholas A., M.A., D.Sc., Associate Professor of Anatomy, *The Daniel Baugh Institute of Anatomy, Jefferson Medical College, Philadelphia, Pa.*

Midlo, Charles, M.D., Clinical Assistant in Surgery, Touro Infirmary, *1514 Delachaise St., New Orleans 15, La.*

Miller, James A., A.B., Ph.D., Assistant Professor of Microscopic Anatomy, *Division of Anatomy, College of Medicine, University of Tennessee, Memphis, Tenn.*

Miller, Roland E., B.S., M.S., Ph.D., Assistant Professor of Anatomy, *Bowman Gray School of Medicine, Winston-Salem, N. C.*

Miller, Ruth Alexander, A.B., Ph.D., Assistant Professor of Anatomy, *Johns Hopkins Medical School, Baltimore 5, Md.*

Miller, Ruth N., A.B., M.D., *7703 West Chester Pike, Upper Darby, Pa.*

Miller, Shirley P., A.B., A.M., Ph.D., Assistant Professor of Anatomy, *Anatomical Institute, University of Minnesota, Minneapolis, Minn.*

Minckler, Jeff, A.B., M.A., Ph.D., M.D., Assistant Professor of Pathology, University of Oregon Medical School, *3205 S. W. Patton Road, Portland 1, Oregon.*

Montagu, M. F. Ashley, Ph.D., Associate Professor of Anatomy, *Hahnemann Medical School and Hospital, Philadelphia, Pa.*

Moody, Robert Orton, B.S., M.D., Emeritus Professor of Anatomy, *University of California, 2826 Garber St., Berkeley, Calif.*

MOORE, CARL R., A.M., Ph.D., Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago, Ill.*

MORGAN, CHARLES F., A.B., Ph.D., Assistant Professor of Pharmacology and Materia Medica, *Georgetown University School of Medicine, Washington, D. C.*

MORGAN, LAWRENCE ONIS, A.B., A.M., Ph.D., Associate Professor of Anatomy, *University of Cincinnati, College of Medicine, Eden Ave., Cincinnati, O.*

MORISON, ROBERT S., A.B., M.D., Assistant Director for the Medical Sciences, *The Rockefeller Foundation, 49 West 49th St., New York 20, N. Y.*

MORRILL, CHARLES V., A.M., Ph.D., Associate Professor of Anatomy, *Cornell University Medical College, 1300 York Ave., New York City.*

MORTENSEN, OTTO A., M.S., M.D., Professor of Anatomy, *University of Wisconsin, 401 Science Hall, Madison 5, Wis.*

MORTON, DUDLEY JOY, M.D., Associate Clinical Professor, Department of Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York City.*

MORTON, THOMAS H., Ph.D., Professor of Biology, *Niagara University, Niagara University, N. Y.*

MOSSMAN, HARLAND W., Ph.D., Associate Professor of Anatomy, *Science Hall, University of Wisconsin, Madison, Wis.*

MOYER, ELIZABETH K., A.B., M.A., Ph.D., Assistant Professor of Anatomy, *School of Medicine, Temple University, Broad and Ontario Sts., Philadelphia 40, Pa.*

MYERS, BURTON D., A.M., M.D., Professor of Anatomy (retired), Indiana University School of Medicine, *424 N. Walnut St., Bloomington, Ind.*

MYERS, J. ARTHUR, M.S., Ph.D., M.D., Professor of Preventive Medicine and Chief of Chest Clinic, *University of Minnesota Medical School, Minneapolis, Minn.*

NELSON, ROSE C., B.A., M.A., Ph.D., *35 Cochran Ave., Ft. Thomas, Ky.*

NELSON, WARREN O., A.B., Sc.M., Ph.D., *Department of Anatomy, State University of Iowa, College of Medicine, Iowa City, Iowa.*

NICHOLAS, JOHN SPANGLER, M.S., Ph.D., Sterling Professor of Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

NICOLL, PAUL ANDREW, A.B., Ph.D., Assistant Professor of Physiology, *Indiana University, Bloomington, Indiana.*

NIELSON, PAUL E., M.S., Ph.D., *Illinois Research Hospital, 1853 West Polk St., Chicago 12, Ill.*

NIGRELLI, ROSS F., B.S., M.S., Ph.D., Pathologist, New York Aquarium, *New York Zoologist Society, Bronx Park, Bronx, N. Y.*

NOBACK, CHARLES ROBERT, B.S., M.S., Ph.D., Assistant Professor of Anatomy, *Long Island College of Medicine, 350 Henry St., Brooklyn 2, N. Y.*

NOBACK, GUSTAVE J., B.S., M.A., Ph.D., *70–05 Groton St., Forest Hills, Long Island, N. Y.*

NONIDEZ, JOSÉ F., M.S., Sc.D., Professor of Anatomy, Cornell University Medical College, *1300 York Ave., New York City.*

NORRIS, H. W., A.B., Sc.D., Research Professor of Zoology, *Grinnell College, Grinnell, Ia.* (Deceased).

OBENCHAIN, JEANETTE B., Ph.D., Research Associate, Department of Anatomy, *The University of Chicago, Chicago, Ill.*

ODIORNE, JOSEPH M., B.S., M.A., Ph.D., Assistant Professor of Anatomy, *Georgetown University School of Medicine, Washington, 7, D. C.*

O'DONOGHUE, CHARLES H., D.Sc., F.Z.S., F.R.S.E., Professor of Zoology, *University of Reading, Reading, England.*

O'LEARY, JAMES L., Ph.D., M.D., Associate Professor of Neuroanatomy and Assistant Professor of Neurology, *Washington University School of Medicine, St. Louis, Mo.*

OPPENHEIMER, JANE M., Ph.D., Assistant Professor of Biology, *Bryn Mawr College, Bryn Mawr, Pa.*

OSBORN, CLINTON M., B.A., M.A., Ph.D., Associate Professor of Anatomy, *College of Medicine, Ohio State University, Columbus, O.*

OSTERUD, HJALMER L., A.B., A.M., Ph.D., Professor of Anatomy, *Medical College of Virginia, Richmond, Va.*

OVERHOLSER, MILTON D., A.B., A.M., Ph.D., M.D., Professor of Anatomy and Chairman of Department, *University of Missouri, Columbia, Mo.*

PAFF, GEORGE H., M.A., Ph.D., Associate Professor of Anatomy, *Long Island College of Medicine, 350 Henry St., Brookliyn, N. Y.*

PALAY, SANFORD L., M.D., 1st Lt. Medical Corps, A.U.S., *10806 Orville Ave., Cleveland 6, Ohio.*

PALMER, CARROLL E., M.A., Ph.D., M.D., Senior Surgeon, *U. S. Public Health Service, Bethesda 14, Md.*

PALMER, DWIGHT M., B.Sc., M.D., Associate Professor of Anatomy and Neuropsychiatry, *Ohio State University, Columbus, O.*

PANKRATZ, DAVID S., A.B., A.M., Ph.D., M.D., Professor of Anatomy, *University of Mississippi, Box 15, University, Miss.*

PAPANICOLAOU, GEORGE N., Ph.D., M.D., Associate Professor of Anatomy, *Cornell University Medical College, 1300 York Ave., New York City.*

PAPEZ, JAMES WENCESLAS, B.A., M.D., Professor of Anatomy, *Cornell University, Ithaca, N. Y.*

PARKER, ALICE E., Ph.D., Instructor in Anatomy, *University of Colorado School of Medicine, Denver, Colo.*

PARKER, GEORGE HOWARD, D.Sc., Professor of Zoology, Emeritus, *Harvard University, 16 Berkeley St., Cambridge 38, Mass.*

PARKER, RAYMOND C., Ph.D., Research Associate, Connaught Laboratories; Lecturer, School of Hygiene, *University of Toronto, Toronto, Canada.*

PARKES, A. S., D. S., F.R.S., Member, *National Institute for Medical Research, Hampstead, London, N.W. 3, England.*

PARSHLEY, MARY STEARNS, A.B., M.A., Ph.D., Research Associate, Department of Pathology, *College of Physicians and Surgeons, Columbia University, New York 32, N. Y.*

PATEK, PAUL R., B.A., Ph.D., Associate Professor of Anatomy, *School of Medicine, University of Southern California, Los Angeles 7, Calif.*

PATTEN, BRADLEY MERRILL, A.M., Ph.D. (Second Vice-Pres. '34–'36), Professor of Anatomy, *University of Michigan, Ann Arbor, Mich.*

PATTERSON, JOHN THOMAS, B.S., Ph.D., Professor of Zoology, *University of Texas, Austin, Tex.*

PEARSON, ANTHONY A., M.A., Ph.D., Associate Professor of Anatomy, *Baylor University, College of Medicine, Houston 1, Tex.*

PEELE, TALMAGE LEE, A.B., M.D., Assistant Professor of Anatomy, *Duke University Medical School, Durham, N. C.*

PENCHARZ, RICHARD I., Ph.D., Research Associate, Zoology Department, University of California, *Rancheria Rd., Kentfield, Calif.*

PERKINS, ORMAN C., A.B., A.M., M.D., F.A.C.P., Professor of Neurology, Long Island College of Medicine, *829 Carroll St., Brooklyn, N. Y.*

PFEIFFER, CARROLL A., Ph.D., Assistant Professor of Anatomy, *Yale University School of Medicine, 333 Cedar St., New Haven, Conn.*

PHELPS, DORIS H., A.B., M.A., Ph.D., Research Associate in Gynecology and Obstetrics, *Vanderbilt University School of Medicine, Nashville, Tenn.*

PHILLIPS, HOWARD MITCHELL, B.S., M.A., Ph.D., Professor of Biology, *Emory University, Georgia.*

PIATT, JEAN, B.S., M.A., Ph.D., Assistant Professor of Anatomy, *School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.*

PICK, JOSEPH, M.D., Associate Professor of Anatomy, *New York University, College of Medicine, 477 First Ave., New York City.*

PIERSOL, WILLIAM HUNTER, B.A., M.B., *103 Hudson Drive, Toronto, Canada.*

PINCUS, GREGORY, B.S., M.S., Sc.D., Director of Laboratories, Worcester Foundation for Experimental Biology, *222 Maple Ave., Shrewsbury, Mass.*

PLAGGE, JAMES C., S.B., Ph.D., Assistant Professor of Anatomy, Department of Anatomy, College of Medicine, University of Illinois, *1853 W. Polk St., Chicago 12, Ill.*

PLISKE, EDWARD C., Ph.D., Instructor in Anatomy, *Medical School, University of North Carolina, Chapel Hill, N. C.*

POHLMAN, AUGUSTUS G., M.D., (Ex. Com. '32–'36), Associate Clinical Professor of Otolaryngology, University of Southern California, *4056 Farmouth Dr., Los Angeles 27, Calif.*

POLYAK, STEPHEN, M.D., Professor of Anatomy, Department of Anatomy, *The University of Chicago, Chicago, Ill.*

POMERAT, CHARLES M., A.B., A.M., Ph.D., Professor of Anatomy, *University of Texas, Medical Branch, Galveston, Tex.*

POMERAT, GERARD ROLAND, A.B., A.M., Ph.D., Instructor in Biology, *Harvard University, Cambridge, Mass.*

POYNTER, CHARLES W. M., B.S., M.D., (Ex. Com. '19–'22), Professor of Anatomy, and Dean of the College of Medicine, University of Nebraska, *Forty-second and Dewey Ave., Omaha, Neb.*

PRATT, JEAN PAUL, M.D., Surgeon in Charge of Divisions of Gynecology and Obstetrics, *Henry Ford Hospital, Detroit, Mich.*

PRYOR, JOSEPH WILLIAM, M.D., Emeritus Professor of Anatomy and Physiology, University of Kentucky, *417 West Second St., Lexington, Ky.*

PUCKETT, WILLIAM O., M.A., Ph.D., Assistant Professor of Biology, *Princeton University, Princeton, N. J.*

QUIRING, D. P., M.A., Ph.D., Associate Professor of Biology, Western Reserve University; Head of Anatomy Division, Cleveland Clinic Foundation, *2249 Demington Dr., Cleveland Heights, O.*

RAMSAY, ANDREW J., A.B., Ph.D., Associate Professor of Histology and Embryology, *Daniel Baugh Institute of Anatomy, Jefferson Medical College, Philadelphia 7, Pa.*

Rasmussen, Andrew T., Ph.D., (Ex. Com. '34–'38), Professor of Neurology, *Institute of Anatomy, University of Minnesota, Minneapolis, Minn.*

Rasmussen, Grant Litster, Ph.D., Associate Professor of Anatomy, *University of Buffalo School of Medicine, 24 High Street, Buffalo, N. Y.*

Rawles, Mary E., A.B., S.M., Ph.D., Research Associate (Embryology), *Department of Biology, Johns Hopkins University, Baltimore 18, Md.*

Rawlinson, Herbert E., M.D., M.Sc., Ph.D., Associate Professor of Anatomy, *University of Alberta, Edmonton, Alberta, Canada.*

Reagan, Franklin P., Ph.D., Lecturer in Anatomy, Hospitals Centre, Medical School, *The University, Edmund St., Birmingham, England.*

Reece, Ralph P., B.Sc., M.Sc., Ph.D., Assistant Professor of Dairy Husbandry, *New Jersey Agricultural Experiment Station, New Brunswick, N. J.*

Reed, Adrian F., A.B., M.A., Ph.D., M.D., Associate Professor of Gross Anatomy, *Tulane University Medical School, New Orleans 15, La.*

Reese, John D., A.B., M.A., M.D., Assistant Professor of Anatomy, *University of California, Berkeley, Calif.*

Reinhardt, William O., A.B., M.D., Instructor and Assistant Professor of Anatomy, *University of California, Berkeley, Calif.*

Revell, Daniel Graisberry, A.B., M.B., Professor Emeritus of Anatomy, University of Alberta, *9105 112 St., South Edmonton, Alberta, Canada.*

Reynolds, Samuel R. M., M.A., Ph.D., Research Associate, *Department of Embryology, Carnegie Institution of Washington, Wolfe and Madison Sts., Baltimore, Md.*

Rhinehart, D. A., A.M., M.D., Professor of Applied Anatomy, University of Arkansas, *7200 W. Markham, Little Rock, Ark.*

Rhines, Ruth, B.S., M.S., Ph.D., Instructor in Anatomy, *Northwestern University Medical School, 303 E. Chicago Ave., Chicago, Ill.*

Rice, Edward Loranus, A.B., Ph.D., Sc.D., Professor Emeritus of Zoölogy, *Ohio Wesleyan University, 265 N. Sandusky St., Delaware, O.*

Richardson, Dorothy, A.B., Ph.D., Associate Professor of Zoology, *Connecticut College, New London, Conn.*

Richter, Kenneth M., B.A., M.A., Ph.D., Assistant Professor of Histology and Embryology, *University of Oklahoma, School of Medicine, Oklahoma City 4, Okla.*

Rienhoff, William F., Jr., A.B., M.D., Associate Professor of Surgery, Johns Hopkins Hospital, *1201 North Calvert St., Baltimore, Md.*

Riley, Henry A., B.A., A.M., M.D., Professor of Clinical Neurology, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York City.*

Ring, John Robert, A.B., Sc.M., Ph.D., Instructor in Micro-anatomy, *Saint Louis University School of Medicine, 1402 S. Grand Blvd., St. Louis 4, Mo.*

Ringeon, Adolph R., B.A., M.A., Ph.D., Professor of Zoology, *University of Minnesota, Minneapolis, Minn.*

Rioch, David McK., M.D., Director of Research, Chestnut Lodge Sanitarium, *500 W. Montgomery Ave., Rockville, Md.*

Risley, Paul L., B.A., M.S., Ph.D., Associate Professor of Zoology, *Department of Biology, University of Oregon, Eugene, Oregon.*

Roberts, Joseph Thomas, B.A., M.S., M.D., Ph.D., Chief Medical Officer, Gallinger Municipal Hospital; Adjunct Clinical Professor of Medicine, Schools of Medicine of Georgetown University and George Washington University; *Gallinger Municipal Hospital, 19th St. and Massachusetts Ave., S. E., Washington, D. C.*

Robertson, G. Gordon, B.A., B.Sc., Ph.D., Assistant Professor of Anatomy, *Baylor University, College of Medicine, Houston 1, Texas.*

Robinson, Byron L., A.B., M.A., M.D., Dean and Professor of Anatomy, *University of Arkansas Medical School, Little Rock, Ark.*

Rogers, James B., A.M., M.D., Professor of Anatomy, *University of Louisville, School of Medicine, 101 W. Chestnut, Louisville, Ky.*

Rogers, Philip V., Ph.D., Assistant Professor of Biology, *Hamilton College, Clinton, N. Y.*

Rogers, William Mitchell, B.S., Ph.D., Assistant Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 632 West 168th St., New York City.*

Romer, Alfred S., B.A., Ph.D., Professor of Zoölogy and Curator of Vertebrate Palaentology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Roncoroni, Enrique J., M.D., Professor of Anatomy, *Universidad del Litoral, Rosario, Republic of Argentina.*

Ronstrom, G. N., B.S., M.S., M.D., Associate Professor of Anatomy, *Louisiana State University Medical Center, 1542 Tulane Ave., New Orleans, La.*

Roofe, Paul Gibbons, B.S., Ph.D., Professor of Anatomy, *University of Kansas School of Medicine, Department of Anatomy, Lawrence, Kansas.*

Roosen-Runge, Edward C., M.D., Pathologist, *Wrentham State School, Wrentham, Mass.*

Rossman, Isadore, A.B., Ph.D., *383 Springfield Ave., Newark 3, N. J.*

Rubinstein, Hyman S., Ph.G., B.S., M.D., Ph.D., Director of the Alfred Ullman Laboratory for Neuro-psychiatric Research, Sinai Hosp., Balto.; Consultant in Psychiatry, U. S. Dist. Court, Md. District, *2349 Eutaw Place, Baltimore 17, Md.*

Rudnick, Dorothea, Ph.B., Ph.D., Assistant Professor of Biology, *Albertus Magnus College, 700 Prospect St., New Haven, Conn.*

Rugh, Roberts, B.A., M.A., Ph.D., Associate Professor of Biology, New York University, *110 Morningside Drive, New York City.*

Ruth, Edward S., M.D., F.A.C.S., *1680 North Vine St., Hollywood, Calif.*

Ruth, Elbert B., A.B., A.M., Ph.D., Associate Professor of Anatomy, *College of Medicine, Eden Ave., University of Cincinnati, Cincinnati 7, O.*

Ryerson, Dwight L., B.A., M.S., Ph.D., Instructor in Anatomy, *University of Arkansas, School of Medicine, Little Rock, Arkansas.*

Sabin, Florence R., B.S., M.D., Sc.D., LL.D. (Second Vice-Pres. '08–'09, Pres. '24–'26), Member Emeritus of Rockefeller Institute for Medical Research, 66th St. and York Ave., New York City, *1333 East 10th Ave., Denver, Colo.*

Saccomanno, Geno, B.A., M.A., Ph.D., Instructor in Anatomy, *St. Louis University School of Medicine, 1402 South Grand Ave., St. Louis, Mo.*

Salmon, Theodora N., B.A., M.A., Ph.D., Instructor, Department of Biological Sciences, Hunter College; Research Associate, Columbia University, *19 Essex Ave., Montclair, N. J.*

Salsbury, C. R., M.D., C.M., F.R.C.S.(C.), F.R.C.S.(Eng.), *2250 McNeill Ave., Victoria, B. C., Canada.*

Sandstrom, Carl J., Ph.D., Associate Professor of Biology, *New York University, New York City 53.*

Sattler, Dwight G., M.D., Instructor in Anatomy, *State University of Iowa, College of Medicine, Iowa City, Ia.*

Sauer, Mary E., A.B., A.M., Ph.D., Assistant Professor of Anatomy, University of Texas, *1115 Ave. E., Galveston, Tex.*

Saunders, John B. deC. M., M.B., Ch.B., F.R.C.S., Professor of Anatomy, Chairman of Division of Anatomy, *University of California, Berkeley, Calif.*

Saunders, Richard L. deC. H., M.B., Ch.B., M.D., Associate Professor of Anatomy, *Dalhousie University, Halifax, Nova Scotia, Canada.*

Sawin, Paul B., Sc.D., Assistant Professor of Biology, *Arnold Laboratory, Brown University, Providence, R. I.*

Sawyer, Charles H., A.B., Ph.D., Assistant Professor of Anatomy, *Duke University School of Medicine, Durham, N. C.*

Scammon, Richard E., Ph.D., LL.D. (Ex. Com. '23–'26, '35–'39, Vice-Pres. '28–'30), Distinguished Service Professor in the Graduate School, *172 S. E. Bedford St., Minneapolis, Minn.*

Schadewald, Melvin, B.S., M.S., Ph.D., Associate Professor of Anatomy, *School of Medicine, University of South Dakota, Vermillion, S. Dak.*

Schaeffer, Bobb, A.B., A.M., Ph.D., Assistant Curator of Fossil Vertebrates, *The American Museum of Natural History, Central Park West at 79th St., New York 24, N. Y.*

Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D. (Ex. Com. '24–'29, Vice-Pres. '42–'43; Acting Pres. '43–'46), Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy, Jefferson Medical College, *4634 Spruce St., Philadelphia 4, Pa.*

Scharrer, Ernst, M.D., Ph.D., Assistant Professor of Anatomy, *School of Medicine, Western Reserve University, 2109 Adelbert Rd., Cleveland 6, O.*

Schmidt, Ida Genther, B.A., M.A., Ph.D., Assistant Professor of Anatomy, *University of Cincinnati, College of Medicine, Eden Ave., Concinnati, O.*

Schoemaker, Daniel M., B.S., M.D., Professor of Anatomy, School of Medicine, St. Louis University, *1402 S. Grand Ave., St. Louis, Mo.*

Schooley, J. P., Ph.D., Associate Professor of Anatomy, *New York Medical College, New York 29, N. Y.*

Schotté, Oscar E., Lic. Sc. Phys. et Nat., Doct. Sc. Nat., Geneva, Switzerland, Professor of Biology, *Amherst College, Amherst, Mass.*

Schour, Isaac, B.S., D.D.S., M.S., Ph.D., Professor of Histology and Head of the Department, University of Illinois College of Dentistry, *808 South Wood St., Chicago, Ill.*

Schultz, Adolph H., Ph.D., Associate Professor of Physical Anthropology, Department of Anatomy, *Johns Hopkins Medical School, Baltimore, Md.*

Schwartz, Henry G., M.D., Instructor in Neurological Surgery, Washington University Medical School, *Barnes Hospital, St. Louis, Mo.*

Schweizer, Malvina, Ph.D., Assistant Professor of Biology, Washington Square College, New York University, *50 West 67th St., New York City.*

Schwind, Joseph L., A.B., A.M., Ph.D., Francis Brunning Professor of Anatomy, and Chairman of the Department, *College of Medicine, University of Cincinnati, Eden Ave., Cincinnati, Ohio.*

Scott, Gordon H., A.B., A.M., Ph.D., Professor of Anatomy and Chairman of Department of Anatomy, *Wayne University College of Medicine, Detroit 26, Michigan.*

Scott, John W., A.M., Ph.D., Professor of Zoology and Research Parasitologist, University of Wyoming, *1409 Garfield St., Laramie, Wyo.*

Sedwick, H. Jobe, D.D.S., *Elderton, Pa.*

Seib, George A., A.B., M.D., Assistant Professor of Anatomy, Washington University, *2323 Lafayette Ave., St. Louis, Mo.*

Selling, Laurence, A.B., M.D., Professor of Medicine, University of Oregon Medical School, *6005 S. W. Hamilton St., Portland 5, Ore.*

Selye, Hans, M.D., Ph.D., D.Sc., F.R.S.(C.), Professor and Director of the Institute of Experimental Medicine and Surgery, *University of Montreal, Montreal, Canada.*

Sensenig, E. Carl, B.A., M.S., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, Tulane University School of Medicine, New Orleans 15, La.*

Severinghaus, Aura E., B.S., M.A., Ph.D., Associate Dean and Associate Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York 32, N. Y.*

Shaner, Ralph Faust, Ph.D., Professor of Anatomy, *University of Alberta, Edmonton, Alberta, Canada.*

Shanklin, William M., Ph.D., Professor of Histology and Neuroanatomy, *Johns Hopkins Medical School, Baltimore, Md.*

Shapiro, Harry H., D.M.D., Assistant Professor of Anatomy, *Columbia University, 630 West 168th St., New York City.*

Sharp, Clayton, M.D., Retired. *414 Powells Lane, Westbury, N. Y.*

Sheehan, Donal, M.D., D.Sc., M.R.C.S., Professor of Anatomy and Director of the Anatomical Laboratories, *New York University, College of Medicine, 477 First Ave., New York City.*

Sheinin, John J., B.S., M.S., Ph.D., M.B., Dean and Chairman Department of Anatomy, *Chicago Medical School, 710 South Wolcott Ave., Chicago, Ill.*

Shields, Randolph T., A.B., M.D., Assistant Professor of Pathology, *Pathological Institute, University of Tennessee, Memphis 3, Tennessee.*

Shryock, E. Harold, B.S., M.A., M.D., Associate Professor of Anatomy, *College of Medical Evangelists, 115 N. San Bernardino St., Loma Linda, Calif.*

Shumway, Waldo, A.B., A.M., Ph.D., Professor of Zoölogy, *University of Illinois, Urbana, Ill.*

Sicher, Harry, M.D., Professor of Anatomy and Histology, *Loyola University School of Dentistry, 706 South Wolcott Avenue, Chicago, Illinois.*

Simer, Parke H., B.Ed., M.A., Ph.D., Associate Professor of Anatomy, *University of Illinois, 1853 W. Polk St., Chicago, Ill.*

Simkins, Cleveland S., A.B., Ph.D., Head, Department of Anatomy, *Creighton Medical School, Omaha, Neb.*

Simpson, Miriam Elizabeth, A.B., M.A., Ph.D., M.D., Professor of Anatomy, Division of Anatomy and Institute of Experimental Biology, *University of California, 1133 Euclid Ave., Berkeley 4, Calif.*

Simpson, William L., B.S., Ph.D., Research Associate, Department of Anatomy, Washington University and The Barnard Free Skin and Cancer Hospital, *Washington University, St. Louis, Mo.*

Sinclair, John G., B.S., M.S., Ph.D., Chairman, Department of Anatomy, and Professor of Histology-Embryology, *University of Texas, Medical Branch, Galveston, Tex.*

Singer, Edward, M.D., Associate Director, St. Clare's Hospital, *111 East 61st St., New York City.*

Singer, Marcus, B.S., A.M., Ph.D., Instructor in Anatomy, *Harvard Medical School, Boston, Mass.*

Smelser, George K., Ph.D., Assistant Professor of Anatomy, *Columbia University College of Physicians and Surgeons, 630 West 168th St., New York City.*

Smith, Carlton G., B.A., M.Sc., M.D., Ph.D., Assistant Professor of Anatomy, *University of Toronto, Toronto 5, Canada.*

Smith, Christianna, A.M., Ph.D., Professor of Zoölogy, *Mount Holyoke College, South Hadley, Mass.*

Smith, David T., A.B., M.D., Associate Professor of Medicine and Professor of Bacteriology, Duke University, *Duke Hospital, Durham, N. C.*

Smith, George Milton, A.B., M.D., Research Associate in Anatomy, Yale University School of Medicine, *Pine Orchard, Conn.*

Smith, Philip Edward, M.S., Ph.D., (Ex. Com. '34–'38), (Pres. '40–'42), Professor of Anatomy, College of Physicians and Surgeons, Columbia University, *632 West 168th St., New York City.*

Smith, Wilbur Cleveland, B.A., M.D., Sc.D., *Tulane University, New Orleans, La.*

Smith, Wilbur K., A.B., M.D., Associate Professor of Anatomy, *University of Rochester School of Medicine and Dentistry, Rochester, N. Y.*

Snider, Ray S., Ph.D., Instructor in Physiology, *The Johns Hopkins University, School of Medicine, Baltimore, Md.*

Snodgrass, L. E., A.B., M.D., Instructor in Surgery, Medical Department, University of Pennsylvania, *7903 Oxford Ave., Philadelphia, Pa.*

Snook, Theodore, Ph.D., Assistant Professor of Anatomy, *Syracuse University, College of Medicine, Syracuse, N. Y.*

Snyder, Franklin Faust, M.D., Associate Professor of Anatomy and Obstetrics, *Harvard Medical School, 25 Shattuck St., Boston, Mass.*

Solnitzky, Othmar, A.B., A.M., Ph.D., M.D., Professor and Director of the Department of Anatomy, Georgetown University, School of Medicine; Director, Georgetown University Brain Research Institute, *3900 Reservoir Rd., Washington, D. C.*

Sosa, Julio Maria, M.D., Head, Division of Histological Research; Assistant Professor of Histology and Embryology, Neurological Institute, School of Medicine, University of the Republic, *P. Vazquez y Vega 1111, Montevideo, Uraguay.*

Spector, Benjamin, M.D., Professor of Anatomy, *Tufts College Medical School, 416 Huntington Ave., Boston, Mass.*

Speidel, Carl Caskey, Ph.B., Ph.D., Sc.D. (Ex. Com. '40–), Professor of Anatomy, *Medical Department, University of Virginia, Charlottesville, Va.*

SPOFFORD, WALTER R., B.S., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, Vanderbilt Medical School, Nashville 4, Tennessee.*

SPRAGUE, JAMES MATHER, A.B., A.M., Ph.D., Instructor in Anatomy, *Johns Hopkins Medical School, Baltimore, Md.*

SPRATT, NELSON, T., A.B., Ph.D., Instructor in Biology; Research Assistant in Embryology, *Department of Biology, Johns Hopkins University, Baltimore, Maryland.*

STARK, MARY B., Ph.B., M.A., Ph.D., Sc.D., Professor of Histology and Embryology, *New York Medical College and Flower Hospital, Fifth Ave. and 105th St., New York City.*

STEIN, KATHRYN F., M.S., Ph.D., Assistant Professor of Zoölogy, *Mount Holyoke College, South Hadley, Mass.*

STILWELL, E. FRANCES, A.B., A.M., Ph.D., Associate Professor of Anatomy, *Woman's Medical College of Pennsylvania, Philadelphia, Pa.*

STONE, LEON STANSFIELD, Ph.B., Ph.D., Bronson Professor of Comparative Anatomy, *Yale University School of Medicine, 333 Cedar St., New Haven, Conn.*

STOPFORD, JOHN SEBASTIAN B., M.D., Sc.D., D.Sc., F.R.S., Vice-Chancellor and Professor of Experimental Neurology, *University of Manchester, Manchester, England.*

STOWELL, ROBERT E., A.B., M.D., Ph.D., Assistant Professor of Pathology, Washington University School of Medicine; Research Associate, Barnard Free Skin and Cancer Hospital, *Department of Pathology, Washington University School of Medicine, St. Louis 10, Mo.*

STRAUS, WILLIAM L., JR., A.B., Ph.D., Associate Professor of Anatomy, *Johns Hopkins Medical School, Baltimore 5, Md.*

STREETER, GEORGE L., A.M., M.D., Sc.D., LL.D., (Ex. Com. '18–'20, Pres. '26–'28), Research Associate, *Carnegie Institution of Washington, Johns Hopkins Medical School, 710 N. Washington St., Baltimore, Md.*

STRÖER, W. F. H., Ph.D., Holland.

STROMSTEN, FRANK ALBERT, B.S., M.S., D.Sc., Associate Professor of Zoölogy, State University of Iowa, *318 Zoology Building, Iowa City, Ia.*

STRONG, LEON H., A.B., B.S., Ph.D., Assistant Professor of Anatomy, Medical School, University of Michigan, *4502 E. Medical Building, University of Michigan, Ann Arbor, Mich.*

STRONG, OLIVER S., A.B., A.M., Ph.D., Professor Emeritus of Neurology and Neurohistology, Columbia University, *200 Retreat Ave., Hartford 2, Conn.*

STRONG, REUBEN MYRON, A.B., A.M., Ph.D., (Ex. Com. '16–'19), Professor of Anatomy, *Loyola University School of Medicine, 706 South Wolcott Ave., Chicago, Ill.*

STULTZ, WALTER A., B.A., Ph.D., Associate Professor of Anatomy, *University of Vermont, College of Medicine, Burlington, Vt.*

STUNKARD, H. W., B.S., A.M., Ph.D., Sc.D., Professor of Biology, *New York University, University Heights, New York City.*

SULLIVAN, WALTER EDWARD, A.M., Ph.D., (Ex. Com. '41–), Professor of Anatomy, University of Wisconsin, *Science Hall, Madison, Wis.*

SWANK, ROY LAVER, Ph.D., M.D., (In national service).

Swenson, E. A., A.B., A.M., Ph.D., address unknown.

Swift, Charles H., M.D., Ph.D., Associate Professor of Anatomy, *The University of Chicago, Chicago, Ill.*

Swingle, W. W., A.B., A.M., Ph.D., Professor of Biology, *Princeton University, Princeton, N. J.*

Swinyard, C. A., M.S., Ph.D., Professor of Anatomy, *School of Medicine, University of Utah, Salt Lake City, Utah.*

Sykes, George F., Ph.B., M.A., Assistant Professor of Anatomy, Tufts College Medical School, *416 Huntington Ave., Boston, Mass.*

Szepsenwol, Josel, M.D., Knight Fellow, Department of Anatomy, *Yale University, School of Medicine, 333 Cedar St., New Haven, Conn.*

Tallmadge, G. Kasten, A.M., M.D., Ph.D., Assistant Professor of Anatomy, Marquette University School of Medicine, *561 North Fifteenth Street, Milwaukee 3, Wisconsin.*

Taylor, A. Cecil, B.A., M.A., Ph.D., Assistant Professor of Anatomy, *College of Dentistry, New York University, 477 First Avenue, New York 16, N. Y.*

Teitelbaum, Harry A., B.S., M.D., Ph.D., Assistant in Neurology, The Johns Hopkins University Medical School; Assistant in Neurology and Instructor in Anatomy, University of Maryland School of Medicine, *1411 Eutaw Pl., Baltimore, Md.*

Telford, Ira Rockwood, A.B., M.A., Ph.D., Assistant Professor in Anatomy, *George Washington University Medical School, 1335 H St., N. W., Washington 5, D. C.*

Terni, Tullio, M.D., Professor of Anatomy, *The University of Padua, Padua, Italy.*

Terrill, Clair Elman, B.S., Ph.D., Animal Husbandman (Physiologist and Geneticist), *U. S. Western Sheep Breeding Laboratory, Dubois, Idaho.*

Terry, Robert James, A.B., M.D., (Ex. Com. '08–'12, '21–'24), Professor of Anatomy, Emeritus, *Washington University School of Medicine, St. Louis, Mo.*

Thomas, Thurlo B., S.B., M.A., Ph.D., Assistant Professor of Anatomy, *University of Texas, Medical Branch, Galveston, Texas.*

Thompson, Ian Maclaren, B.Sc., M.B., Ch.B., Professor of Anatomy and Director of the Department of Anatomy, *Medical College, University of Manitoba, Winnipeg, Canada.*

Thomson, Stewart Craig, A.B., M.S., M.D., Assistant Professor of Anatomy, Assistant Dean, *Loyola University School of Medicine, 706 S. Wolcott Ave., Chicago, Ill.*

Thorkelson, Jacob, M.D., Member of Congress; MC-V (S) USNR, *National Hotel, Butte, Mont.*

Thuringer, Joseph M., M.D., Professor of Histology and Embryology, University of Oklahoma, School of Medicine, *801 N. E. 13th St., Oklahoma City, Okla.*

Tobin, Charles E., Ph.D., Associate Professor of Anatomy, *The University of Rochester, School of Medicine and Dentistry, 260 Crittenden Blvd., Rochester, N. Y.*

Tompkins, Edna H., M.D., Research Associate, (Associate Professor) *Laboratory of Applied Physiology, Yale University, New Haven, Conn.*

Tower, Sarah Sheldon, B.S., Ph.D., M.D., Associate in Anatomy, *Johns Hopkins Medical School, Baltimore, Md.*

TRACY, HENRY C., A.B., A.M., Ph.D., Professor of Anatomy, *University of Kansas, Lawrence, Kan.*

TROTTER, MILDRED, A.B., M.S., Ph.D., Associate Professor of Anatomy, *Washington University School of Medicine, St. Louis, Mo.*

TRUEX, RAYMOND C., M.S., Ph.D., Associate Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York City.*

TRUSCOTT, BASIL LIONEL, B.A., M.A., M.S., Ph.D., Medical Student, *Box 260, Yale University School of Medicine, New Haven, Conn.*

TUNTURI, ARCHIE ROBERT, A.B., M.S., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, University of Oregon Medical School, Portland, Oregon.*

TURNER, CHARLES W., B.S., A.M., Ph.D., Professor of Dairy Husbandry, *University of Missouri, Columbia, Mo.*

TURNER, C. DONNELL, Ph.D., Assistant Professor of Zoology, *Northwestern University, 105 Locy Hall, Evanston, Ill.*

TURNER, CLARENCE L., M.A., Ph.D., Professor of Zoology, *Northwestern University, Evanston, Ill.*

TURNER, ROBERT S., A.B., Ph.D., Assistant Professor of Anatomy, *Stanford University, Stanford University, Calif.*

UHLENHUTH, EDUARD, Ph.D., Professor of Anatomy, *University of Maryland Medical School, Baltimore, Md.*

UOTILA, UNTO U., M.D., Research Fellow in Anatomy, Harvard Medical School, Boston, Mass. (1939); Docent in Microscopical Anatomy, *Helsinki University, Siltavuorenpenger 20, Helsinki, Finland.*

VAN CAMPENHOUT, ERNEST, M.D., Ph.D., Professor of Anatomy, Histology and Embryology, *Université de Louvain, Louvain, Belgium.*

VAN CLEAVE, C. D., Ph.D., Associate Professor of Anatomy, *School of Medicine, University of North Carolina, Chapel Hill, N. C.*

VAN DER HORST, C. J., D.Sc., Professor, Head of the Department of Zoölogy, *University of the Witwatersrand, Johannesburg, So. Africa.*

VAN DYKE, JOHN H., A.B., M.S., Ph.D., Assistant Professor of Anatomy, *Washington University School of Medicine, St. Louis 10, Mo.*

VAN WAGENEN, GERTRUDE, A.B., Ph.D., Research Associate in Obstetrics and Gynecology, Associate Professor's rank, Yale University, *New Haven Hospital, New Haven, Conn.*

VENABLE, JOHN HEINZ, B.S., M.D., Associate Professor of Anatomy, *Emory University, Emory, Ga.*

VICARI, EMELIA, A.B., A.M., Research Fellow, *Roscoe B. Jackson Laboratory, Bar Harbor, Me.*

VON BONIN, GERHARDT, M.D., Professor of Anatomy, University of Illinois, College of Medicine, *1853 West Polk St., Chicago, Ill.*

VONDERAHE, A. R., A.B., B.Sc., M.D., Associate Professor of Anatomy, *University of Cincinnati, College of Medicine, Eden Ave., Cincinnati, O.*

WAITE, FREDERIK CLAYTON, A.M., Ph.D., Emeritus Professor of Histology and Embryology, Western Reserve University School of Medicine, *144 Locust St., Dover, N. H.*

WALLER, WILLIAM H., Ph.D., Associate Professor of Anatomy, *University of Georgia, Medical School, Augusta, Ga.*

WALLIN, IVAN E., M.A., D.Sc., Professor of Anatomy and Head of Department, *Secretary of Faculty, University of Colorado, School of Medicine, Denver, Colo.*

WALLS, G. L., Sc.D., Research Associate, *Scientific Bureau, Bausch and Lomb Optical Co., Rochester, N. Y.*

WALMSLEY, THOMAS, M.D., F.R.S.E., Professor of Anatomy, *Queen's University of Belfast, Belfast, Ireland.*

WARBRITTON, VIRGENE, Ph.D., (Mrs. Fred Kavanagh), *New York Botanical Gardens, New York 58, N. Y.*

WARD, JAMES W., Ph.D., M.D., Associate Professor of Anatomy, *Vanderbilt University Medical School, Nashville, Tenn.*

WARREN, A. EMERSON, B.A., Ph.D., Professor of Zoology, *McMaster University, Hamilton, Ontario, Canada.*

WARREN, CHARLES O., A.B., Ph.D., M.D., Assistant Professor of Anatomy and Physiology, *Cornell University Medical College, 1300 York Ave., New York City.*

WASHBURN, SHERWOOD L., A.B., Ph.D., Assistant Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 623 West 168th St., New York City.*

WASSERMANN, FRIEDRICH, M.D., Professor of Anatomy, *Dept. of Anatomy, The University of Chicago, Chicago, Ill.*

WANTERMAN, ALLYN J., A.B., A.M., Ph.D., Associate Professor of Biology, *Williams College, Williamstown, Mass.*

WATKINS, RICHARD WATKIN, S.B., M.D., Assistant Professor of Otolaryngology, Rush Medical College, *5608 Kenwood Ave., Chicago 37, Ill.*

WATT, JAMES CRAWFORD, M.A., M.D., Professor of Anatomy, *University of Toronto, Toronto, Canada.*

WEATHERFORD, HAROLD LORRAINE, A.B., A.M., Ph.D., Assistant Professor of Anatomy, *Harvard Medical School, Boston, Mass.*

WEAVER, HARRY M., A.B., M.Sc., Ph.D., Assistant to the Medical Director, The National Foundation for Infantile Paralysis, *120 Broadway, New York 5, N. Y.*

WEBB, RICHARD L., A.B., M.S., Ph.D., Professor of Anatomy, *College of Medicine, University of Illinois* (on leave, '45–'46). Visiting Professor of Anatomy, *Washington University School of Medicine, St. Louis, Mo.*

WEBER, NEAL A., B.A., M.S., M.A., Ph.D., Associate Professor of Anatomy, Medical School, University of North Dakota, *University Station, Grand Forks, North Dakota.*

WEED, LEWIS HILL, A.B., A.M., M.D., Sc.D., LL.D., (Ex. Com. '20–'21, '35–'39, Sec.-Treas. '22–'30), Professor of Anatomy and Director, *Johns Hopkins Medical School, Baltimore, Md.*

WEICHERT, CHARLES KIPP, B.S., M.S., Ph.D., Professor of Zoology, *University of Cincinnati, Cincinnati 21, O.*

WEIDENREICH, FRANZ, Dr. Med., Honorary Director, Cenozoic Laboratory, Peiping Union Medical College, Peking, China; Research Associate, *American Museum of Natural History, New York 24, N. Y.*

Weinstein, Bernard, B.S., M.D., M.S., Assistant Professor of Gross Anatomy and Gynecology, Tulane University of Louisiana, *1441 Delachaise St., New Orleans 6, La.*

Weiss, Paul Alfred, Ph.D., Professor of Zoology, *The University of Chicago, Chicago, Ill.*

Weissenberg, Richard, M.D., *54 Orange Street, Waltham, Mass.*

Wells, Lemen J., A.M., Ph.D., Associate Professor of Anatomy, *Institute of Anatomy, University of Minnesota, Minneapolis, Minn.*

Weston, Jean K., M.S., Ph.D., M.D., Professor of Anatomy, Temple University School of Medicine, *Broad at Ontario St., Philadelphia, Pa.*

Wheatley, Max D., M.A., Ph.D., Research Biologist in Charge of Animal Research, *Illinois Neurosychiatric Institute, 912 S. Wood St., Chicago 12, Ill.*

Wheeler, Theodora, A.B., M.D., Clinical Associate in Neurology, *Rush Medical College, 1758 W. Harrison St., Chicago, Ill.*

Whitaker, Wayne L., A.B., A.M., Ph.D., Assistant Professor of Anatomy, *Department of Anatomy, University of Michigan, Ann Arbor, Mich.*

Whiteside-Harvel, Beatrice, B.A., Ph.D., Voluntary Research Worker, *Department of Histology, University of Geneva School of Medicine, Geneva, Switzerland.*

Wieman, H. L., Ph.D., Professor of Zoology, *University of Cincinnati, Cincinnati, O.*

Wierda, Jay L., Ph.D., Assistant Professor of Anatomy, *Scohol of Medicine, University of Pennsylvania, Philadelphia 4, Pa.*

Willard, William A., A.M., Ph.D., Professor of Anatomy, University of Nebraska, *College of Medicine, Forty-second St. and Dewey Ave., Omaha, Neb.*

Williams, George Dee, A.B., A.M., M.D., Ph.D., Assistant Professor of Anatomy, *Washington University School of Medicine, St. Louis, Mo.*

Williams, Roy Griffin, A.M., M.D., Associate Professor of Anatomy, *University of Pennsylvania School of Medicine, Philadelphia 4, Pa.*

Williams, Stephen Culver, Ph.D., Assistant Professor of Anatomy, *Medical Department, University of Pennsylvania, Philadelphia 4, Pa.*

Williams, W. Lane, B.S., M.A., Ph.D., Assistant Professor of Anatomy, *University of Minnesota, The Medical School, Minneapolis 14, Minnesota.*

Willier, Benjamin Harrison, Ph.D., D.Sc., Henry Walters Professor of Zoology and Chairman of Department of Biology, *Johns Hopkins University, Charles and 34th Sts., Baltimore 18, Md.*

Wilson, James G., B.A., M.A., Ph.D., Assistant Professor of Anatomy, *The University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.*

Wimsatt, William Abell, A.B., Ph.D., Assistant Professor of Zoology, *Department of Zoology, Cornell University, Ithaca, N. Y.*

Windle, William F., B.S., M.S., Ph.D., Professor of Anatomy, *University of Washington School of Medicine, Seattle 5, Washington.*

Wislocki, George B., A.B., M.D., A.M. (hon.), (Ex. Com. '37–'41), James Stillman Professor of Comparative Anatomy, *Harvard Medical School, Boston, Mass.*

Witschi, Emil, Ph.D., Professor of Zoology, *State University of Iowa, Iowa City, Ia.*

Woerner, Charles A., Ph.D., M.D., Instructor in Anatomy, *New York Medical College, 1 East 105th St., New York 29, N. Y.*

Wolfe, J. M., B.S., Ph.D., Professor of Anatomy, and Director of the Department, *Albany Medical College, Albany 3, N. Y.*

Woodburne, Russell T., M.A., Ph.D., Associate Professor of Anatomy, *University of Michigan, Ann Arbor, Mich.*

Woodman, Alice S., M.D., *Hotel Statler, Boston 17, Mass.*

Worcester, John Locke, M.D., Professor of Anatomy, University of Washington, *5211 Twenty-first Ave., N. E., Seattle, Wash.*

Wright, Philip Lincoln, B.S., M.S., Ph.D., Assistant Professor of Zoology, *Montana State University, Missoula, Montana.*

Wright, Walter H., D.D.S., B.S., M.S., Ph.D., Professor of Anatomy and Prosthetic Dentistry, School of Dentistry, University of Pittsburgh, *1306 Penn Ave., Wilkinsburg Sta., Pittsburgh, Pa.*

Yeakel, Eleanor H., A.B., M.A., Ph.D., Fellow, *The Wistar Institute of Anatomy and Biology, Philadelphia 4, Pa.*

Yntema, C. L., Ph.D., Assistant Professor of Anatomy, *Cornell University Medical College, New York City.*

Yoffey, J. M., M.Sc., M.D., F.R.C.S., Professor of Anatomy, *The University, Bristol, Great Britain.*

Young, M. Wharton, M.D., Ph.D., Associate Professor of Anatomy, *Howard University, Washington, D. C.*

Young, William C., Ph.D., Associate Professor of Biology, *Cedar Crest College, Allentown, Pa.*

Youngstrom, Karl A., A.B., M.A., Ph.D., M.D., Interne Cleveland Clinic Foundation, *Cleveland Clinic, Cleveland 6, Ohio.*

Zacharias, Leona Ruth, B.A., M.A., Ph.D., Instructor in Anatomy, *Columbia University, College of Physicians and Surgeons, New York City.*

Zechel, Gustav, M.D., F.A.C.S., Assistant Professor in Anatomy and Surgery, University of Illinois College of Medicine, *1853 W. Polk St., Chicago, Ill.*

Zeit, Walter, B.S., M.S., Ph.D., Associate Professor of Anatomy, *Marquette University School of Medicine, 561 North 15th St., Milwaukee 3, Wis.*

Zimmermann, Arnold, Lic.Sc., D.Sc., (Geneva, Switzerland), Professor of Anatomy, University of Illinois College of Medicine, *1853 W. Polk St., Chicago, Ill.*

Zweifach, Benjamin William, B.S., M.S., Ph.D., Research Associate in Biology, *Department of Medicine, Cornell University Medical College, 1300 York Ave., New York 21, N. Y.*

Zwemer, Raymund L., A.B., Ph.D., Executive Director, Inter-departmental Committee on Cultural and Scientific Cooperation and Special Assistant to the Director, Office of International Information and Cultural Affairs, *5003 Battery Lane, Bethesda, Md.*

THE ANATOMY AND FUNCTION OF THE FORELEG IN SALAMANDER LOCOMOTION[1]

F. GAYNOR EVANS
Department of Anatomy, Wayne University College of Medicine,
Detroit, Michigan

SIX FIGURES

INTRODUCTION

Much work has been done on the mechanics of locomotion and of movement in man and in other mammals (Marey, 1874; Haycraft, '00; Fick, '11; Gregory, '12; Braus, '29; Steindler, '35; and others), but relatively little has been done on Amphibia. Nauck ('24) and Schaeffer ('41), in their studies of the hind leg in Amphibia and in Reptilia, have given it some consideration but the best account is in a recent and excellent paper by Gray ('44).

Some investigators (Gray, '39; Gray and Lissmann, '40), using spinal preparations, have approached the problems of amphibian locomotion from the viewpoint of the relationship of the central nervous system to the locomotor muscles. Most of the attention has been focused on the role of the hind legs in locomotion, the part played by the forelegs being either ignored or considered in a very general way.

Consequently, it seems to be generally believed that the forelegs merely support the front part of the body so that the hind legs can do their work of propulsion. As this is only partially true the present study was undertaken with the view of trying to determine something about the role of the forelegs in salamander locomotion as well as the anatomical and mechanical factors involved.

---

[1] Assisted in part by a Research Grant from the American Association for the Advancement of Science.

MATERIAL AND METHODS

With these things in mind locomotion in Triturus granulosus, T. torosus, T. viridescens and Ambystoma opacum was studied by direct observation and by slow motion pictures. The pictures were taken of T. granulosus and consisted of 100 feet of 16-mm. Eastman Super XX film taken with a Cine-Kodak Special camera equipped with a 1-inch lens. The first 60 feet of film were taken at a distance of 32 inches from the subject and the last 40 at a distance of 36 inches. A stop of f. 5.6 and a speed of 64 frames per second were used.

In addition, still pictures of T. torosus were taken with a Speed Graphic camera equipped with a Zeiss Ikon lens. Eastman Super XX film, a stop of f. 8 and a speed of 1/150 of a second were used with the subject 24 inches from the lens.

In order to have a side, a dorsal, and a ventral view of the animal simultaneously, a wooden frame was built to hold a horizontal glass plate and two plate glass mirrors. Each mirror was placed at a 45° angle to the horizontal glass plate on which the animals walked while being photographed. A strip of white cardboard about 2 inches wide and the same length as the horizontal plate served as a background to enhance the profile of the animal in side view. The camera was placed with the lens at the same level as the horizontal plate. The complete apparatus in operation is illustrated in figure 1, top, while figure 1, bottom, is an enlarged view of the subject.

ACKNOWLEDGMENTS

For the slow motion pictures I am indebted to Mr. Thane L. Bierwert, Staff Photographer of the American Museum of Natural History, while the still pictures were taken by Mr. Thomas Hoffmaster of the Art Department, University of Maryland School of Medicine.

Living specimens to study as well as material for dissection I owe to the generosity of Mr. Charles M. Bogert, American Museum of Natural History, Dr. Doris M. Cochran, United

States National Museum, and Dr. Eduard Uhlenhuth, Department of Gross Anatomy, University of Maryland School of Medicine. I also want to express my appreciation to Doctor Uhlenhuth for the facilities he provided for me while I was a member of his department.

For figures 5 and 6, I am indebted to Miss Evelyn J. Erickson of the Department of Medical Illustration, Wayne University College of Medicine.

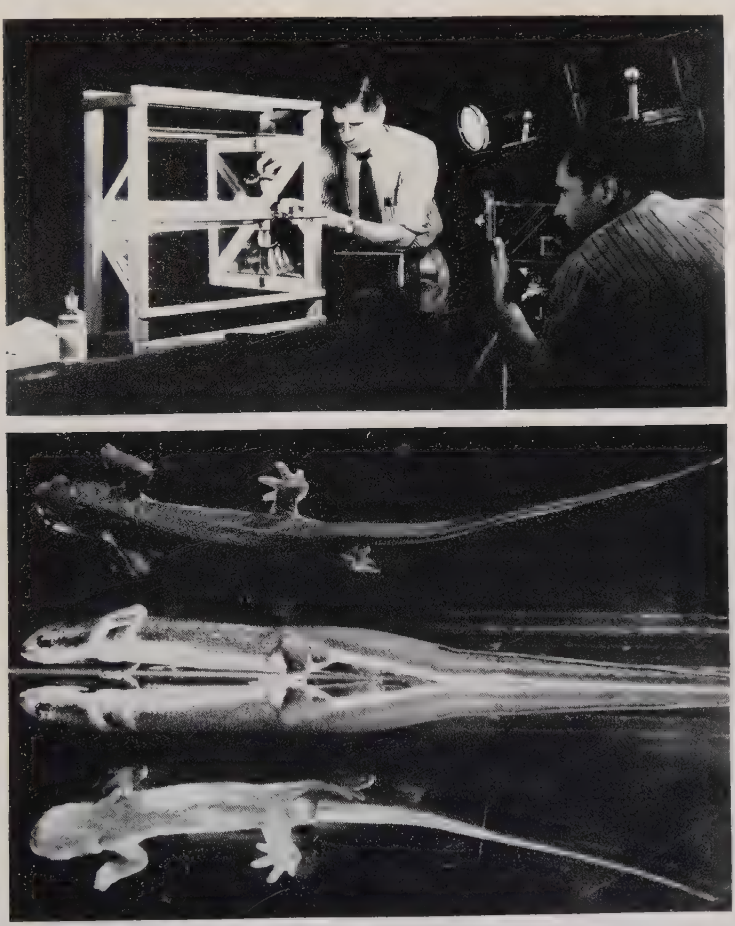


Fig. 1 A. Photographic apparatus.
B. Enlarged view of the subject.

DISCUSSION OF FINDINGS

Two separate and distinct types of terrestrial locomotion, depending upon the speed of the animal, are to be found in salamanders. When the animal, e.g., Ambystoma opacum, is frightened it can move quite rapidly, for a short distance, by means of a violent wriggling of the body over the ground. This rapid type locomotion is produced by the alternate contraction of the well-developed axial musculature of the trunk so that the animal practically "swims" over the surface of the ground. The mucus secreted by the belly skin aids in this movement by reducing the friction between the body and the ground, while the legs play only a slightly less important part than they do in actual swimming.

The minor role of the legs in rapid locomotion is clearly illustrated when the animal is forced to move over smoked kymograph paper. Examination of the records made by Ambystoma opacum (fig. 2 A) and Triturus viridescens (fig. 3 A) during rapid locomotion shows no definite footprints whatever. Instead, the main part of the record consists of large irregular smear marks and a more or less undulating line caused by drag of the body and the tail, respectively, on the paper. On each side of these marks are a series of finer curved lines. Observation of the animal during rapid locomotion shows that the finer lines are made by the tips of the digits which are dragged over the surface of the paper during the recovery phase. The position of these marks shows that the legs, although playing little active part in rapid locomotion, move in the same sequence as they would if the animal were actually walking.

In contrast to this kind of record is that (figs. 2 B and 3 B) obtained for the same two species when the animals are undisturbed and moving in a slow deliberate manner. In this second type of locomotion lateral undulations of the trunk, produced by the axial musculature, still occur but the legs now play an important locomotory role. Thus, examination of the record reveals that even in a heavy-bodied type such as Ambystoma opacum (fig. 2 B) the trunk and tail are com-

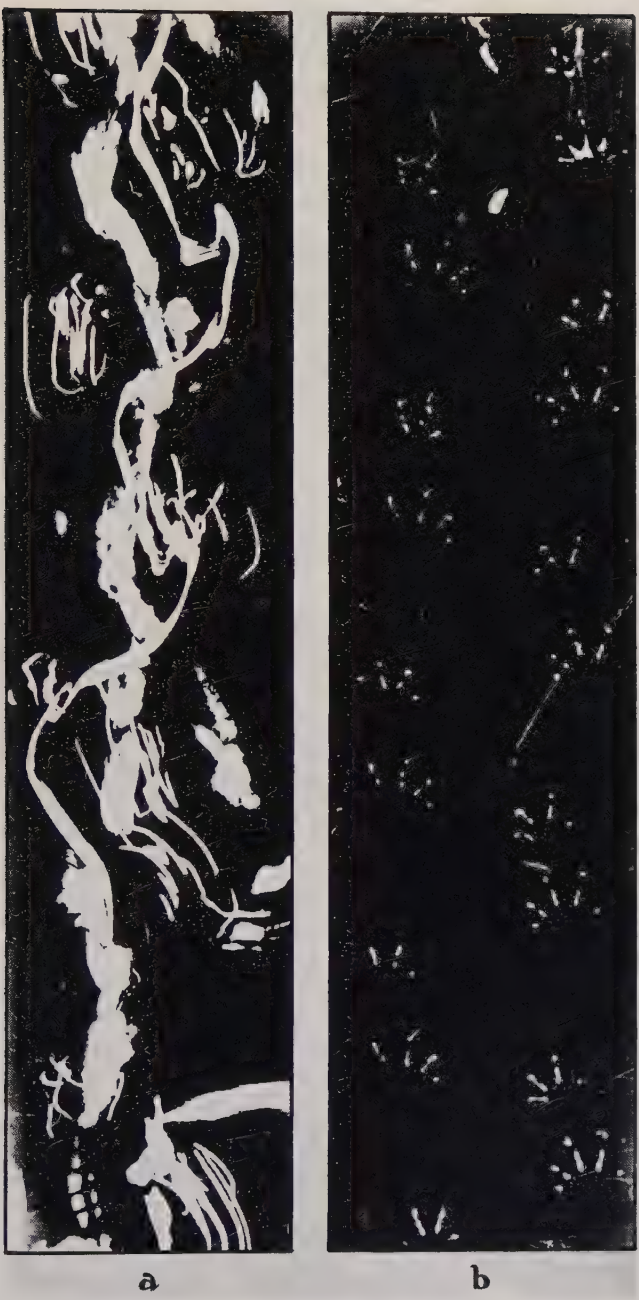


Fig. 2 Kymograph record of Ambystoma opacum.
A. Rapid locomotion. B. Slow locomotion.

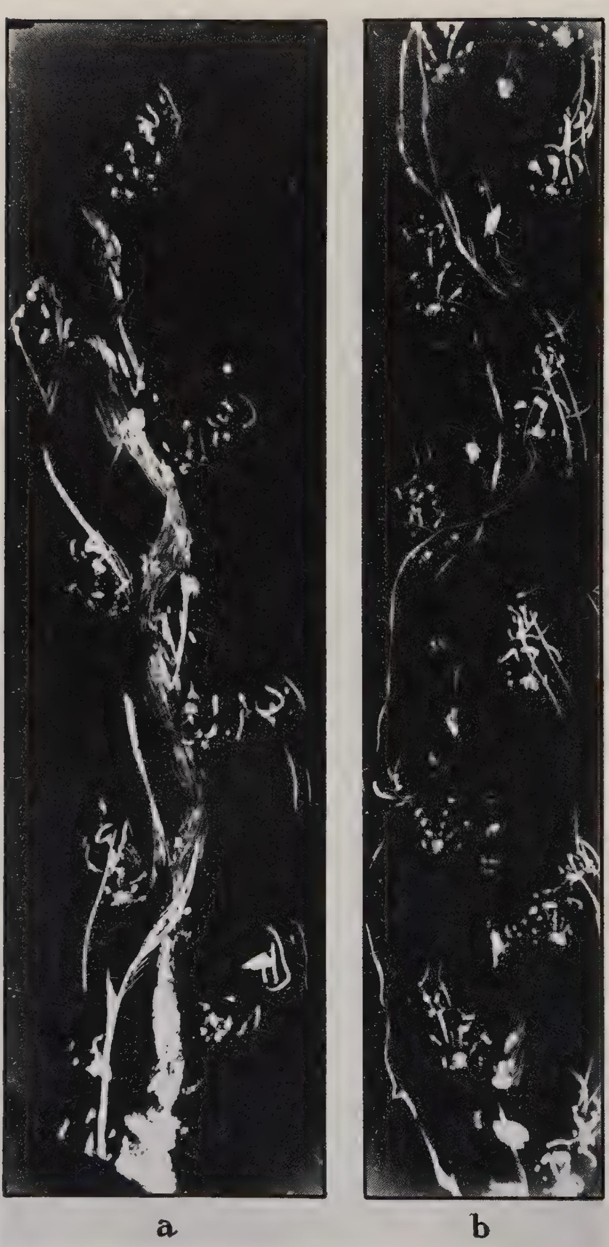


Fig. 3 Kymograph record of Triturus viridescens.
A. Rapid locomotion. B. Slow locomotion.

pletely raised from the paper. The body is also entirely off the ground in Triturus viridescens (fig. 3 B) although the tip of the long tail shows a slight drag. In this slow deliberate type of movement a clear record of footprints, without any body drag, is obtained for Ambystoma (fig. 2 B) showing that the legs are strong enough to support the body off the ground and that they are the chief factor in locomotion. In the case of Triturus (fig. 3 B) the record of the footprints, because of the light weight of the animal, is not as clear. However, because of the adverse mechanical position of the legs, which spread out laterally from the body instead of being beneath it as in mammals, the animal tires rapidly and is not able to support the body off the substratum for any great length of time. It is with the function of the forelegs in this second slow type of locomotion that the present study is concerned. Five different stages in this type of locomotion are illustrated in figure 4.

Movements of the foreleg during locomotion

Movements of the forelegs are essentially the same in Triturus granulosus, studied with the aid of slow motion pictures, as in the other species employed in this investigation. In all of them, for purposes of analysis, the movements may be considered as occurring in two phases: (1) propulsive and (2) recovery. The propulsive phase consists of a backward movement of the humerus accompanied by flexion of the forearm and carpo-metacarpal regions while during the recovery phase the opposite movements take place.

Propulsive phase. During its backward swing the humerus moves in a horizontal plane, with its distal end at a slightly higher level than the proximal end, and at the conclusion of the phase it is at an angle of about 38° with the long axis of the body. Simultaneously with this movement the elbow and the carpo-metacarpal joints are undergoing flexion while the flexor muscles of the manus are pressing the digits against the ground, thus increasing the friction and preventing backward slipping. The end of the propulsive phase is marked by the lifting of the manus from the ground, the digits (Triturus granulosus) being raised from the substratum in the following sequence: IV, I, III, II. During most of this phase the elbow and carpal regions are flexed although towards the end of the phase they are extended somewhat.

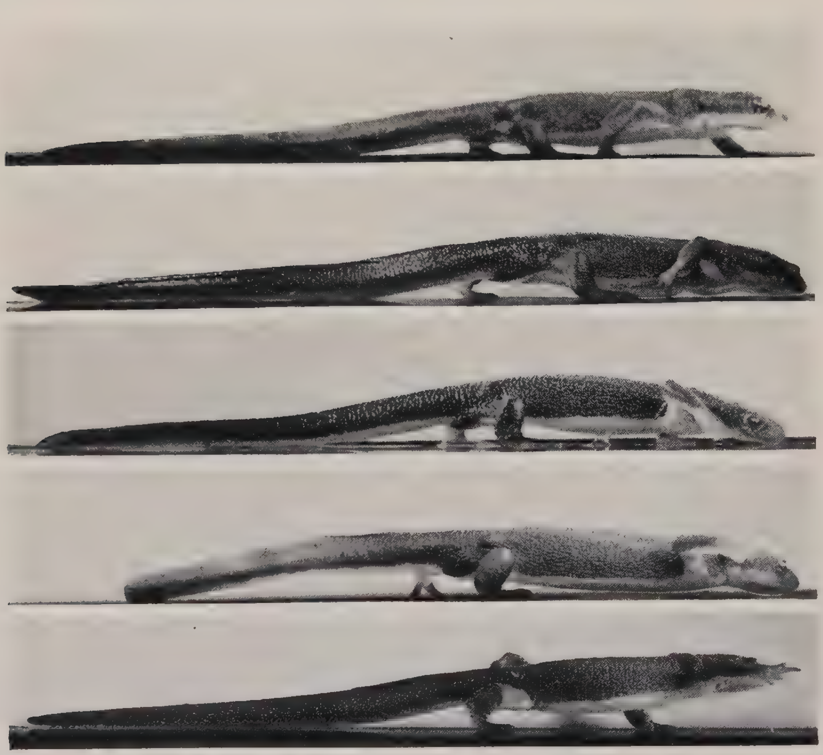


Fig. 4 Five stages during slow locomotion of Triturus torosus.

Recovery phase. The raising of the digits from the substratum initiates the recovery phase during which the humerus swings forward, still in the horizontal plane, to a position of about a 45° angle to the long axis of the body. The elbow is flexed all during the recovery phase and the manus and digits strongly so at the beginning of it. However, towards the end of the phase the manus and digits begin to unroll or extend. The placing of the manus on the substratum again marks the end of the recovery and the beginning of the propulsive phase. Rarely, at this time, do the tips of the digits extend beyond the end of the snout. The entire recovery phase is quite similar to the overhand stroke in swimming except that the salamander elbow is never fully extended.

The preceding description is the normal sequence of events of foreleg movements during slow locomotion when the animal is unexcited. These are essentially similar to those Schaeffer ('41, pp. 412–414) described for the hind legs of salamanders except that the foreleg, during recovery, is not raised as high above the ground as is the hind leg; and the palm, in contrast to the sole, is never directed laterally. Under normal conditions the animals may remain inactive for relatively long periods, simply lying on the ground with the legs in whatever position they happened to be at the time.

Snyder ('04, p. 285) found essentially the same two methods of locomotion in Batrachoseps stating that for leisurely movements only the legs were used. However, in contrast to the results found in the present study, he states that during this type of locomotion the body was "dragged passively along."

From the preceding description of the movements of the foreleg when the animal is walking it is evident that the arm is the propulsive element of the limb. It is the driving lever which, swinging back and forth in a horizontal plane, propels the body forward. The forearm acts as a secondary lever transmitting the propulsive force from the arm to the point of resistance, the substratum. It also supports the weight of the body which is transmitted along the radial margin of the forearm through the manus to the ground. The manus represents the expanded base of this weight-supporting column (forearm) and adds little to the propulsive stroke, serving chiefly as a friction pad to prevent backward slipping. It is aided in this function by the weight of the body and the flexors of the digits which keep them firmly pressed against the ground.

Additional information on the role of the manus is provided by the record made when the animal is walking (fig. 2 B and fig. 3 B). Close examination of the record reveals that only the digits make a print. At no time is the palm of the manus in contact with the paper which indicates that the weight of the body is supported by the digits.

When the animal is at rest is the only time the palm of the hand leaves an imprint and even then only a small area at the base of the first two digits is recorded. This, together with the fact that these two digits leave the clearest marks, suggests that they support the most weight.

In both methods of urodele locomotion lateral undulations of the trunk occur although they are much more violent in rapid movement. In walking the effect of this, with respect to the forelegs, is to swing the body forward on the side where the leg is in the recovery phase and hence raised off the ground. During this forward swing, the opposite leg, on the side where the axial muscles are contracting, is in contact with the ground and is undergoing propulsive movements. The limb is thus not only a propulsive organ but a prop or pivot around which the body is swung. During this process the propulsive leg is on the concave side of the body while the leg in the recovery phase is on the convex side of the body and thus has the length of its stride increased. The absence of fusion between the opposite halves of the pectoral girdle, so that they have considerable independent mobility, is another factor tending to increase the length of stride. Schaeffer ('41, p. 412) reported similar events occurring with reference to the hind legs.

As Gray ('39, p. 48) has pointed out, the sequence of flexor leg movements in the lower terrestrial tetrapods follows a basic pattern which is, beginning with flexion of the right foreleg, (1) right foreleg, (2) left hind leg, (3) left foreleg, and (4) right hind leg. He also states that "as the speed of movement increases, the time interval between the two members of each diagonal pair of limbs decreases until the movement of both members is almost simultaneous." In contrast to mammals, such as the horse, this was the only variation in gait that he noted in urodeles.

Strength of the forelegs

In order to secure some idea of the pulling power of the forelegs the following simple experiments were made. In three specimens of Ambystoma opacum the hind legs were immobilized by binding them to the sides of the body with a strip of paper, and the resulting effect on locomotion was observed. All specimens were still able to accomplish fair forward progress by active walking movements of the forelegs accompanied by vigorous lateral undulations of the trunk and tail. When the posterior part of the body was raised from the ground forward locomotion approached the normal condition.

In four other specimens the front legs were immobilized in the same manner while the hind legs were left free. This had a decidedly more deleterious effect on locomotion, forward progress being extremely slight and only attained by laborious use of the hind legs and violent wriggling of the trunk and tail. However, when the fore part of the body was raised from the substratum good forward movement was possible as a result of the walking action of the hind legs.

Thus, the forelegs alone are seen to be capable of pulling the body over the surface of the ground so that fairly good forward progress results. The pushing power of the hind legs alone is only sufficient for very laborious forward movement which, in no case, was as effective as that produced by the forelegs alone. When the hind legs are working alone the direction of the propulsive force is such that it tends to push the body into the ground at the point of contact, while the propulsive force of the forelegs is directed so that it drags the body at the point of contact with the ground. In both cases the legs were aided by the action of the axial musculature.

The unexpected strength of the forelegs and shoulders was further revealed by vertically suspending specimens of Triturus granulosus from the edge of a glass shelf with which only the forelegs were in contact. Each specimen was able to pull the rest of its body up onto the shelf, much the same way as a man would if he were hanging by his hands.

The anatomical basis for the locomotory function of the forelegs

The following description of the anatomy of the salamander shoulder and foreleg is based on a dissection of these regions in Dicamptodon ensatus, the largest species of living land salamander, the specimen dissected being 250 mm long and weighing 51.5 gm. Additional information, particularly with respect to the general function of individual muscles, was obtained by comparison of the condition in Dicamptodon with that in Salamandra as described by Francis ('34).

Whenever, in the subsequent discussion, a muscle is said to act as a lever of a certain class it should be clearly understood that the lever is the skeletal part upon which the muscle acts and not the muscle itself which merely supplies the power for movement.

Pectoral girdle. The pectoral girdle consists of a large, flat cartilaginous coracoid plate with a cartilaginous procoracoid process projecting cranially. Rising dorsally from this is the scapula and a large, expanded suprascapular cartilage. Only the scapula and a small area round the glenoid cavity are ossified. The two coracoid plates overlap and between their caudal parts, but superficial to both, is a thin cartilaginous sternal plate (sternum).

Shoulder joint. This is a well developed enarthrosis permitting quite free movements of the humerus in the horizontal and vertical planes. Rotation, however, is more limited, being restricted by contact of the head of the humerus with the walls of the glenoid cavity. The latter is V-shaped, when seen ventrally, and is directed laterally, thus causing the humerus to be held in a horizontal plane.

The head of the humerus is terminal in position and somewhat elliptical in shape, being more strongly curved in the cranio-caudal direction than in the dorso-ventral one. Thus, the articular surface in the cranio-caudal direction forms the arc of a smaller circle than in the dorso-ventral direction. Consequently, the humerus has a wider range of movement in the horizontal plane than in the vertical plane. The entire shoulder joint is enclosed in a strong capsule which limits the freedom of movement to some extent.

Muscles acting on the shoulder joint (fig. 5). Seven muscles: the latissimus dorsi, the dorsalis scapulae, the pectoralis, the supracoracoideus, the procoraco-humeralis, the coraco-brachialis, and the subscapularis act on the shoulder joint. All of them, except the coraco-brachialis, are large, flat triangular muscles arising from some part of the pectoral girdle of the fascia of the region and inserting on the humerus.

The retractors of the humerus are the latissimus dorsi, the pectoralis, the subscapularis, and the coraco-brachialis. The latissimus arises from the dorsal fascia in close association with the skin, the pectoralis from the fascia of the rectus abdominis, and the subscapularis from the dorsal surface of the procoracoid cartilage. All three muscles insert on the humerus close to the head, the two latter muscles on the ventral and dorsal crests, respectively. Therefore, these three muscles act as Class III levers with a short power arm and their contraction would produce quite rapid movements of the distal end of the humerus. During locomotion the speed of these movements is greatly reduced by contact of the manus with the ground, in which case the body is pulled forward on the same side.

The latissimus is apparently the most efficient as its angle of insertion is greater than that of the other two muscles. Consequently, a large proportion of the force of its contraction is applied to the humerus. The angle of insertion of the pectoralis is small, which would indicate, according to Steindler ('35, p. 65), that in all probability the stabilizing component of its muscle force is greater than the rotatory component. The subscapularis has its mechanical efficiency enhanced by the passage of its muscle fibers around the posterior border of the coracoid, thus increasing their angle of insertion on the humerus. Most of the subscapularis fibers are parallel and pull on the humerus in the same direction. In the latissimus and pectoralis, however, because of their shape, fibers in various parts of the muscle pull on the humerus in different directions.

The coraco-brachialis is a fusiform muscle consisting of two parts (longus and brevis). Mechanically it acts as a Class III lever and should have greater power than the preceding muscles because of its long power arm, the pars longus inserting on the distal end of the humerus at a considerable distance from the head or fulcrum. In addition, because of the distal and proximal insertion of the pars longus and pars brevis, respectively, the force of contraction is applied to the humerus throughout most of its length. The muscle fibers run nearly parallel with each other and most of them exert a similar pull on the humerus. However, because of the small angle of insertion, this muscle would probably also have a strong stabilizing effect.

The procoraco-humeralis, arising from the ventral surface of procoracoid cartilage and inserting on the ventral crest of the humerus, protracts the humerus and rotates it inward so that the pre-axial border of the foreleg and manus are depressed. This action probably occurs chiefly during the recovery phase when the foot is off the ground. However, if the foot were on the ground, the inward rotation of the humerus would aid slightly in propulsion. This muscle also acts as a Class III lever with, because of its insertion close to the humerus head (fulcrum), a short power arm. Its action would produce quite rapid movements of the distal end of the humerus and of the forearm. Its angle of insertion is about 50° indicating that a relatively large proportion of the force of its contraction is applied directly to the humerus. The muscle is flat and triangular in shape but much longer and narrower than either the pectoralis or the supracoracoideus. This increased length provides a greater distance through which the muscle can contract. The mechanical factors mentioned in connection with this muscle probably account for the rapid recovery movements of the fore limb.

The arm is raised in the vertical plane by the dorsalis scapulae, a triangular muscle arising from the dorso-lateral border of the suprascapular cartilage and inserting on the ventral crest of the humerus close to the head. This muscle acts as a Class III lever with a short power arm. Its contraction would produce a rapid movement of the distal end of the humerus, a considerable amount of the force of this contraction, because of the large angle of insertion (about 60°), being applied directly to the bone.

The antagonist of the preceding muscle is the supracoracoideus, a large triangular muscle arising from the ventral surface of the coracoid cartilage and inserting, together with the pectoralis, on the ventral crest of the humerus near the head. It acts as a Class III lever with a short power arm but, because of its low angle of insertion, its stabilizing component is probably greater than its rotational component.

The elbow joint. The elbow joint is of the ginglymus type with a well developed capsule. In the walking position, because of the torsion of the humerus, the ball-shaped capitulum is placed dorsal to the shallow trochlea. Consequently, the joint axis is almost vertical with reference to the horizontal plane. The epiphyses of the bones forming the joint are quite well ossified.

The semilunar notch of the ulna is continuous with the articular surface of the head of the radius, the two bones being so closely appressed that a single, semicircular, concave articular surface is formed.

Flexion, extension, and a slight amount of rotation of the forearm occur at this joint. Supination is prevented by contact of the ulna with the medial condyle of the humerus. The range of these movements is limited by the joint capsule and the action of antagonistic muscles. If the joint capsule is removed and the muscles bisected, the forearm can be completely extended on the arm although this does not occur during locomotion.

The development of an olecranon process and the displacement of the radius lateral to the ulna are factors associated with terrestrial locomotion. Because of these, Lubosch ('10) considers the elbow joint of the Caudata to be of a higher rank than a simple ginglymus type.

Muscles acting on the elbow joint (fig. 5). The muscles acting on the elbow joint are: the anconaeus (''triceps''), the humero-antibrachialis, the extensor carpi ulnaris, the extensor carpi radialis, the flexor carpi ulnaris, the flexor carpi radialis, and the flexor antibrachii ulnaris. All of these muscles are somewhat fusiform in shape and have their fibers running almost parallel. Therefore, practically all the fibers of a muscle pull on the bone in the same direction.

The large powerful ''triceps'' is the most important extensor of the forearm and covers nearly all the posterior and lateral surface of the arm. Its origin is from the scapula, immediately dorsal to the glenoid fossa, and from the shaft of the humerus while its insertion is on the olecranon process close to the elbow joint or fulcrum. Mechanically this muscle acts as a lever of the First Class which because of its short power arm provides for great speed. The moving component of the muscle force is great because of the large angle of insertion.

The other forearm extensors are the extensor carpi ulnaris and the extensor carpi radialis. The latter also extends the manus. Both of these muscles arise on the lateral epicondyle of the humerus and insert on the distal end of the ulna and the radius, respectively, as well as on the carpus. They act as Class III levers with a long power arm. Consequently their contraction would result in greater power than speed of movement. Their angle of insertion is small, probably indicating an important stabilizing function.

The humero-antibrachialis is also a Class III lever, arising on the flexor surface of the humerus and inserting on the proximal end of the radius. Because of the short power arm its contraction produces rapid flexion of the forearm. The angle of insertion is large so that most of the force of contraction is applied directly to movement of the forearm.

Other forearm flexors are the flexor carpi radialis and the flexor carpi ulnaris. The latter also flexes (depresses) the manus. Both muscles arise from the medial epicondyle of the humerus and insert on the distal end of the radius and on the carpals, respectively. As the flexor carpi ulnaris inserts only on the carpals it is not as powerful a forearm flexor as the flexor carpi radialis. Functionally these muscles act as Class III levers. The contraction of the flexor carpi radialis, because of its long power arm, results in powerful but not rapid movement. The flexor carpi ulnaris is only a slight flexor of the forearm, its chief function being to flex or depress the manus. It has a short power arm, inserting on the ulnare and the intermedium close to the ulno-carpal joint or fulcrum. Consequently it would produce rapid but not powerful movements. Both of these muscles have a small angle of insertion indicating they may have a stabilizing function.

Beneath the flexor carpi ulnaris is the flexor antibrachii ulnaris, a short muscle arising on the medial epicondyle of the humerus and inserting on the proximal half of the radius. It retracts the forearm, relative to the body, and thus, if the foot is on the ground, assists in advancing the body. It acts as a Class III lever with a short power arm, thus producing a more rapid movement than the flexor carpi radialis. However, the latter, because it is a longer muscle, can contract through a greater distance. Judging from its small angle of insertion the flexor antibrachii ulnaris may well exercise a stabilizing influence.

Running in a diagonal direction between the radius and ulna, and acting as an elastic ligament to hold them together, is the interosseus antibrachii muscle.

The carpus. The carpus of Dicamptodon consists of eight ossified elements: radiale, intermedium, ulnare, centrale, and four distal carpalia. The distal end of the radius is more expanded than that of the ulna and has a greater share in the formation of the ''proximal transverse carpal'' joint. This joint, formed by the radius and the ulna on one hand and the proximal row of carpals (radiale, intermedium, ulnare) on the other, is the site of flexion and extension movements of the manus as a whole. Flexion has a greater range than extension which is restricted by contact of the radius and ulna with the intermedium. The latter is a large bone which in extension becomes wedged into a notch between the distal end of the radius and the ulna.

The only other place in the carpal region where any appreciable movement can occur is the carpo-metacarpal or ''distal transverse carpal'' joint. Flexion and extension can occur here but the range of movement is much less than in the ''proximal transverse carpal'' joint or in the metacarpal-phalangeal joints. The individual carpal bones are connected with each other by elastic tissue so that the entire carpus is quite supple although the degree of movement between the separate bones is slight. The metacarpal-phalangeal and the interphalangeal joints are of the ginglymus type. The phalangeal formula is 2–2–3–2.

The entire hand is very supple and during locomotion bending movements occur successively at the ''proximal transverse carpal,'' the ''distal transverse carpal,'' and the metacarpal-phalangeal joints. The greatest movements are in the first and the last of the above joints, the hand tending to be rolled forward over the ground during propulsion.

ABBREVIATIONS

AEDP, abductor and extensor digiti primi
AN, anconaeus (''triceps'')
CB, coraco-brachialis
CC, coracoid cartilage
CD, contrahentes digitorum
CLMC, caput longum musculorum contrahentium
DF, dorsal fascia
DS, dorsalis scapulae
EBD, extensores breves digitorum
ECR, extensor carpi radialis
ECU, extensor carpi ulnaris
EDC, extensor digitorum communis
ELDQ, extensor lateralis digiti quarti
EO, external oblique
FAL, flexor accessorius lateralis
FAM + PP, flexor accessorius medialis and pronator profundus
FBP, flexores breves profundi
FBS, flexores breves superficialis
FCP, flexor communis primordialis
FCR, flexor carpi radialis
FCU, flexor carpi ulnaris
HA, humero-antibrachialis
IMC, intermetacarpal
LD, latissimus dorsi
P, pectoralis
PCC, procoracoid cartilage
PH, procoraco-humeralis
SC, supracoracoideus
SSC, suprascapular cartilage
UL, ulna
I, first digit
IV, fourth digit

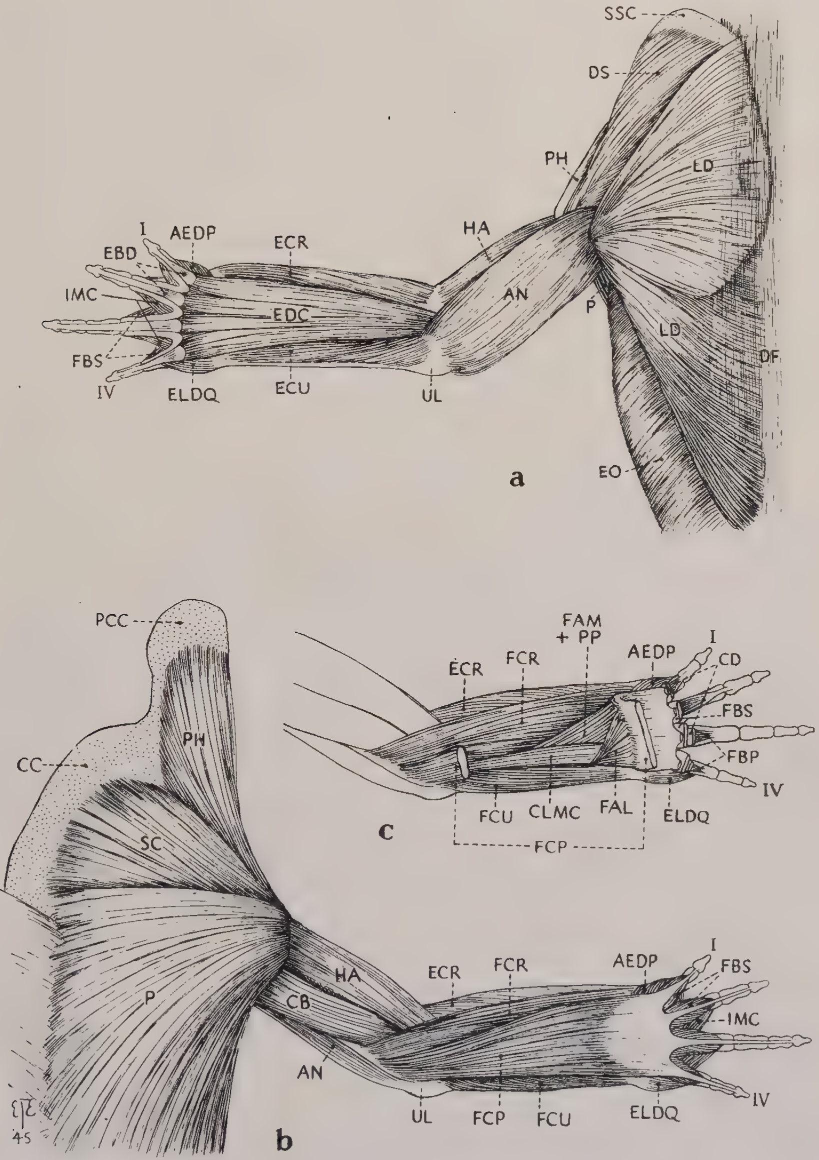

SSC
DS
PH
LD
HA
I
AEDP
EBD
ECR
IMC
EDC
AN
P
LD
DF
FBS
IV
ELDQ
ECU
UL
EO
a
PCC
FAM
+ PP
AEDP
I
CD
ECR
FCR
FBS
FBP
IV
FCU
CLMC
FAL
ELDQ
FCP
c
CC
PH
SC
HA
CB
P
AN
ECR
FCR
AEDP
I
FBS
IMC
IV
UL
FCP
FCU
ELDQ
b

Fig. 5 Muscles of the shoulder and fore limb in Dicamptodon ensatus.
A. Dorsal view of shoulder and extensor muscles of fore limb.
B. Ventral view of shoulder and superficial flexor muscles of fore limb.
C. Ventral view of deep flexor muscles of forearm.

Muscles acting on the manus (fig. 5). The carpus and the manus as a whole is extended chiefly by the extensor digitorum communis (fig. 5 A) assisted by the carpal portions of the extensores carpi radialis and ulnaris. It is a large fleshy muscle arising from the lateral humeral epicondyle and inserting, by means of small tendons, into each side of the base of the metacarpals. In doing so it passes over the carpal and the carpo-metacarpal joints but the range of movements in them is so slight that the extensor digitorum communis may be considered to function as a Class III lever with the fulcrum at the "proximal transverse carpal joint." Because of its short power arm it should have fairly rapid action.

The digits are extended by the extensores breves digitorum which, on superficial inspection, appear as part of the preceding muscle. Closer examination reveals them to be four small muscles arising from the carpalia, beneath the extensor digitorum communis, and inserting, by a long tendon, on the terminal phalanx of each digit. The tendons are held in place by connective tissue and, when the digits are flexed, the direction of tendon pull varies with each segment of a finger. The mechanical efficiency of the muscles is enhanced by the expanded ends of the phalanges which act as pulleys for the tendon passing over them and, in the case of the terminal phalanx, increase its angle of insertion.

Two other short extensors of the digits are the abductor and extensor digiti primi, acting on the first digit, and the extensor lateralis digiti quarti, acting on the fourth digit. The former muscle arises from the ulnare, intermedium, and centrale; the latter from the angle between the ulnare and intermedium. The insertions are on the radial margin of the first metacarpal and the proximal part of the fourth metacarpal, respectively. The function of the abductor and extensor digiti primi is given by its name while the extensor lateralis digiti quarti abducts the fourth digit and assists in extending the hand. Both muscles act as Class III levers with a short power arm.

In the interval between adjacent digits, and visible on both extensor and flexor surfaces of the manus, are the intermetacarpal muscles (three in each manus). These are short triangular muscles running in a proximo-distal direction between adjacent metacarpals. They are adductors of the digits (i.e., the metacarpals), acting as Class III levers with the fulcrum at the carpal-metacarpal joint.

The common flexor of the manus and digits (fig. 5 B) is the flexor communis primordialis, a large flat muscle arising from the medial epicondyle of the humerus and inserting, by means of a long tendon, on the terminal phalanx of each digit. The palmar portion of the muscle is aponeurotic while the remainder is fleshy. The majority of the fibers are longitudinal and parallel in their arrangement, hence, most of the force of contraction would be expended in direct pull on the tendons. The direction of tendon pull itself is changed in each segment of a digit by a small loop of connective tissue which acts as a pulley for the tendon passing through it. These pulleys and the insertion on the terminal phalanx are close to the interphalangeal joints or fulcra and, because of this, the action of the muscle on each segment of a digit may be considered to be that of a Class III lever with a short power arm. This would provide for speed of movement rather than power. The expanded ends of the phalanges slightly increase the angle of the tendon's insertion on the terminal phalanx thus enhancing mechanical efficiency.

Dorsal to the preceding muscle and inserting on its palmar aponeurosis are two small accessory (medialis and lateralis) flexors (fig. 5 C) of the manus. Both of them arise from the distal portion of the ulna, the ulnare and the intermedium. They lie medial and lateral, respectively, to the caput longum musculorum contrahentium (ulnaricarpalis of Wilder) and function as Class III levers with a fairly short power arm. Most of the fibers are parallel and the angle of insertion is small. The more distal portion of the flexor accessorius medialis mass, whose fibers insert on the base of the first two metacarpals, is sometimes considered as a separate muscle, the pronator profundus, whose action is suggested by its name.

The primary flexor of the carpus is the caput longum musculorum contrahentium, a long slender muscle arising from the proximal end of the ulna and inserting on the carpalia close to the "proximal transverse carpal" joint or fulcrum. Mechanically it acts as a Class III lever which, because of the short power arm, should produce rapid movement. The muscle fibers are parallel with a small insertion angle indicating a considerable stabilizing effect on the joint.

The short flexors of the digits, named in order from the superficial to the deep position, are the flexores breves superficialis, the contrahentes digitorum, and the flexores breves profundi. The flexores breves superficialis arise from the dorsal surface of the palmar aponeurosis and insert on the metacarpals near the base of the proximal phalanges. Arising from the carpalia and inserting on the proximal ends of the first phalanges are the musculi contrahentes digitorum, while the angles between the distal carpalia provide the origin for the flexores breves profundi which insert on their respective metacarpals.

All three groups of muscles function as Class III levers with the fulcra for the first and third group being at the "distal transverse carpal" joint while the metacarpal-phalangeal joints represent the fulcra for the middle layer of muscles. The range of movement is small in all three groups because of the shortness of the muscles.

Function of the fore limbs in equilibrium (fig. 6)

In equilibrium in firmisternal amphibians (e.g., Ranidae) the fore limbs can function as transverse struts (Gray, '44) but in arciferous forms such as salamanders, in which the opposite coracoid cartilages overlap, the limbs act as levers moved by the pectoral muscles. Thus, the bilateral compression of the thorax by the laterally displaced limbs is avoided.

The conditions of equilibrium in a salamander, together with some of the mechanical forces involved, are diagrammatically illustrated in figure 6. The limbs are assumed to be equally loaded and equally inclined with reference to the horizontal plane. The principal muscles concerned are the pectoralis and the "triceps" both of which function as antigravity muscles.

The pectoralis adducts the arm, thus rotating the humerus downward and inward so that the proximal end tends to exert an upwardly directed force on the pectoral girdle while the distal end pushes down on the forearm. The "triceps" prevents flexion of the forearm on the arm although its complete extensory effect is inhibited by friction. The rotational effect or moment of each muscle is equal to the tensile strength of the muscle (magnitude of the force) multiplied by the perpendicular distance from the action line of the muscle to the center of rotation of the joint.

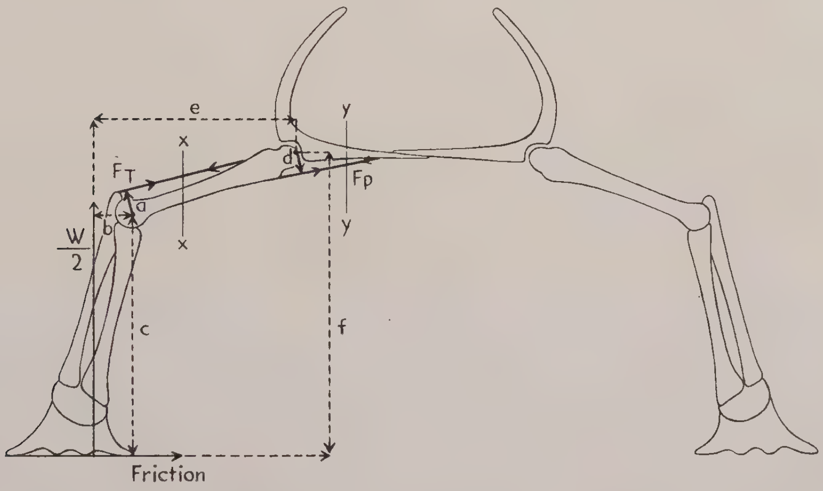


Fig. 6 Diagram of the salamander pectoral girdle and fore limb illustrating the antigravitational functions of the pectoralis and "triceps" (anconaeus) muscles in a state of equilibrium. See text for explanation.

For determining the effect of the "triceps" and pectoralis individually the muscles are assumed to have been cut approximately in half along the lines XX and YY, respectively. Thus, only the portion of the diagram to the left of the lines XX and YY need be considered in dealing with the respective muscles.

In the state of equilibrium the force exerted by the "triceps" may be represented by the formula

$$F_T a + \frac{W}{2} b - Fc = 0$$

where F_T = tension in the "triceps"; $\frac{W}{2}$ = the weight borne by the limb; F = friction; a = the perpendicular distance from the action line of the muscle to the center of rotation of the joint; b and c = the distance from the center of rotation of the joint to the action lines of $\frac{W}{2}$ and F, respectively.

Under similar conditions of equilibrium the force exerted by the pectoralis is

$$F_P d - \frac{W}{2} e + Ff = 0$$

where F_P = tension in the pectoralis; d = the perpendicular distance from the action line of the muscle to the center of rotation of the joint; e and f = the distance from the center of rotation of the joint to the action lines of $\frac{W}{2}$ and F, respectively.

SUMMARY

1. The role of the fore limb during locomotion in four species of salamanders was studied by means of slow motion pictures, kymograph records, and direct observation.
2. A comparative study of the locomotor power of the fore limbs versus the hind limbs was made.
3. Kymograph records show two methods of locomotion: (1) a rapid method during which the animal "swims" over the ground with little assistance from the legs, and (2) a slow method in which the legs are used and the body is raised from the ground.
4. The arm is the propulsive element of the limb, while the forearm is the weight-supporting element which transmits the propulsive force to the ground. The manus is the

expanded base of the weight-supporting column (forearm) and, aside from being a friction pad, adds little to the propulsive stroke.

5. The osteology and myology of the pectoral girdle and limb of Dicamptodon ensatus, together with the joint and muscle mechanics involved, are discussed in detail in order to provide an anatomical basis for explanation of the locomotor role of the salamander fore limb.

6. The functions of the pectoralis and "triceps," as antigravity muscles during the state of equilibrium, and some of the mechanical principles concerned are discussed.

LITERATURE CITED

Braus, H. 1929 Anatomie des Menschen. 3 BB., Julius Springer, Berlin.

Fick, R. 1911 Handbuch der Anatomie und Mechanik der Gelenke. 3 BB., Gustave Fischer, Jena.

Francis, E. T. 1934 The anatomy of the salamander. Oxford Univ. Press.

Gray, J. 1939 Aspects of animal locomotion. Proc. Roy. Soc., Ser. B, vol. 128, pp. 28–62.

——— 1944 Studies in the mechanics of the tetrapod skeleton. J. Exp. Biol., vol. 20, pp. 88–116.

Gray, J., and H. W. Lissmann 1940 Ambulatory reflexes in spinal amphibians. J. Exp. Biol., vol. 17, pp. 237–251.

Gregory, W. K. 1912 Notes on the principles of quadrupedal locomotion. Ann. N. Y. Acad. Sci., vol. 22, pp. 267–294.

Haycraft, J. B. 1900 Animal mechanics. Pp. 228–273 in "Text-book of Physiology" by E. A. Schäfer. Macmillan, N. Y.

Lubosch, W. 1910 Bau und Entstehung der Wirbelturgelenke. Gustave Fischer, Jena.

Marey, E. J. 1874 Animal Mechanism. Appleton and Company, N. Y.

Nauck, E. T. 1924 Die Beziehungen zwischen Beckenstellung und Gliedmassenstellung bei tetrapoden Vertebraten. Morph. Jahr., vol. 53, pp. 1–47.

Schaeffer, B. 1941 The morphological and functional evolution of the tarsus in amphibians and reptiles. Bull. Amer. Mus. Nat. Hist., vol. 78, pp. 395–472.

Snyder, C. D. 1904 Locomotion in Batrachoseps with severed nerve cord. Biol. Bull., vol. 7, pp. 280–288.

Steindler, A. 1935 Mechanics of normal and pathological locomotion in man. Charles C. Thomas, Springfield and Baltimore.

THE RELATION OF HYDROSTATIC CONDITIONS IN THE UTERUS TO THE SIZE AND SHAPE OF THE CONCEPTUS DURING PREGNANCY: A CONCEPT OF UTERINE ACCOMMODATION

S. R. M. REYNOLDS
Department of Embryology, Carnegie Institution of Washington, Baltimore, Maryland

THREE FIGURES

The changes in shape which the uterus undergoes during gestation are common knowledge to those concerned with gynecic physiology. Nevertheless, recent observations on this subject in the course of other studies require that it be given new emphasis. These observations pertain to the relation of the shape and size of a conceptus to the tension of the uterine wall around the fetus, and to the physical factors which govern this relation. Certain functional corollaries are implicit in these considerations. These are concerned with the general problem of uterine growth; the general hemodynamic conditions of blood flow in the uterine wall; and the effects of these conditions of the maternal environment about the fetus upon its growth and welfare.

I. GENERAL CONSIDERATIONS

The accommodation of the conceptus by the uterus follows a pattern which, for convenience, may be divided into three distinct phases. These are:

1. Period of uterine preparation
2. Period of uterine enlargement
3. Period of uterine stretching

The characteristics of each of the above phases are as follows.

Period of uterine preparation. Between the time of conception and the time of nidation, the classical progestational changes first described by Ancel and Bouin ('10) and demonstrated to be related to progesterone by Corner and Allen ('29) take place within the uterus. These have been frequently and well described. It is sufficient for the purpose of the present discussion to note that this time is characterized by mitotic proliferation of the uterine tissues, both in the endometrium and in the myometrium, although the proliferation of glandular elements is best known. No appreciable increase in size (weight or volume) of the uterus takes place. It therefore follows, as has been emphasized before (Reynolds, '39), that this phase is characterized by an increase in the number of component parts of the uterus which are available for subsequent hypertrophy in the next phase of uterine accommodation of the products of conception.

Period of uterine enlargement. This phase commences soon after implantation of the blastocysts. Following nidation, certain grossly discernible changes take place. Within a short period of time, the weight of the gravid uterus alone (i.e., less the products of conception) increases. The curve of uterine enlargement shown in figure 1 indicates the form of the uterine growth curve for the rabbit, as it is at present understood (adapted from Reynolds, '37). Similar data exist for the rat (Siegmund, '30), cow (Hammond, '27), the human (Ivy, '42) and for certain other animals. All show the same sigmoid form in which the period of uterine enlargement lies between two other periods of insignificant increase in size of the uterus. The enlargement which takes place in this second phase is characterized by hypertrophy of tissue elements, without mitosis except where the trophoblast is forming (Reynolds, '39). Certain details of this phase merit special emphasis, however, in the light of the relations to be pointed out below.

At each implantation site, small spheroids are formed by the uterine wall about each conceptus. There is local uterine distention, as shown in figure 1a. This spheroid was drawn from a uterus taken from a rabbit 9½ days after mating. One uterine cornu had been rendered sterile by prior tubal ligation. The other cornu contained three spheroidal implantation sites. The two cornua had the following displacement volumes: non-gravid cornu, $3.9\ \mathrm{cm}^3$; gravid cornu, $4.7\ \mathrm{cm}^3$. The sum of the calculated volumes of the spheroids, using the formula, $V = \frac{4}{3}\pi r^3$, is $0.70\ \mathrm{cm}^3$. Mesial and lateral diameters measurements[1] of the inter-implantation sites were almost exactly equal to those of the non-gravid cornu. Thus it is seen that there is little enlargement, if any, of the uterus up to this time. The fact has been pointed out in previous publications that the non-gravid horn in a unilateral pregnancy does not increase in size until toward the very end of the period of uterine enlargement for the gravid uterus (cf. fig. 1; also Reynolds, '37, '39).

Uterine enlargement during the remainder of the second phase consists essentially of hypertrophy of the tissues around the spheroidal conceptus. This is shown in figure 1, a, b, c, d, and e, which depicts quite exactly the relative change in size of each conceptus site on days 9, 12, 16, 20, 22, 24, and 28 of pregnancy in rabbits in which there are four or five conceptuses in a uterine cornu. Deviations from this rate of change will occur when the number of young in a cornu is larger or smaller than three or four (Reynolds, '39).

Period of uterine stretching. This period of uterine accommodation is associated with a great decrease in the rate of uterine growth, and commencement of the maximum absolute rate of increase in the size of the products of conception. It is, therefore, a time when the products of conception are contained in an ever-increasingly stretched uterus, until the conditions giving rise to the onset of labor supervene. It occurs as a consequence of gradual fetal growth in a lengthwise direction, and an absolute as well as a relative decrease in the amount of fluid about the fetus (cf. Reynolds, '39).

---

[1] Mesial diameter measurements are made by use of a caliper from the mesial margin of the uterus to the opposite or antimesial side of the uterus; lateral diameters are measured at right angles to the mesial-antimesial diameter, at the point of maximum size.

During this time, the conceptus-site is no longer spheroidal or ovoidal. As many observers have noted, it undergoes elongation so that it is cylindroid in shape. In the rabbit, this transition from the spheroid to the elongated form occurs between the twentieth and the twenty-fourth days. Most frequently, it occurs about the twenty-second day. Reference to figure 1 will show that this is the point of transition in the

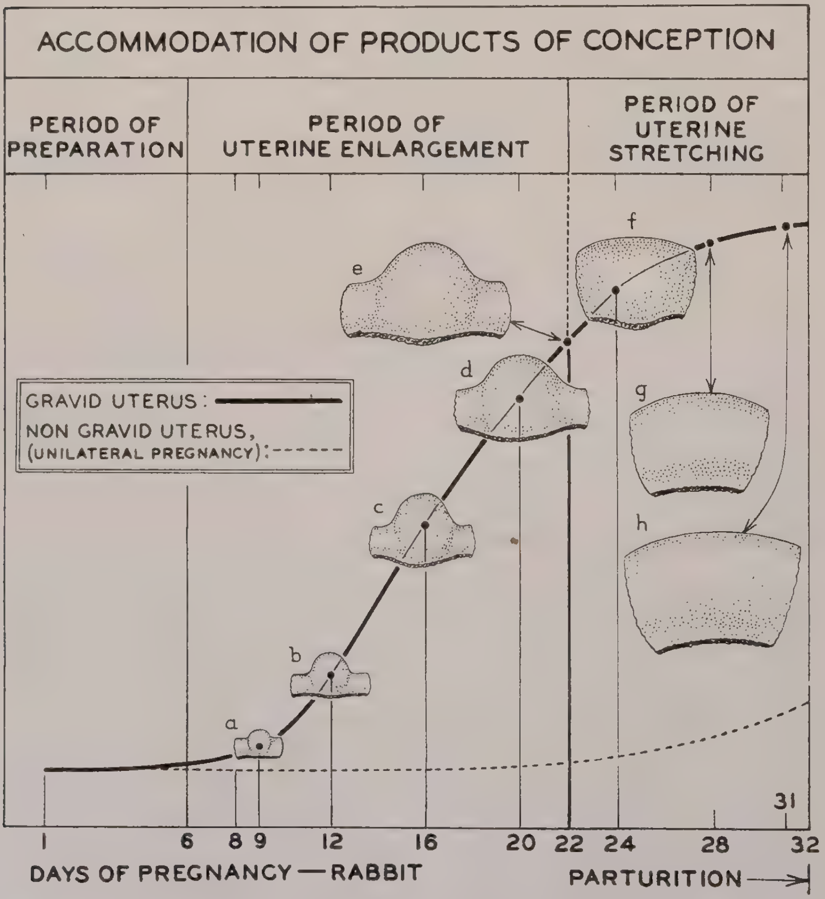

ACCOMMODATION OF PRODUCTS OF CONCEPTION
PERIOD OF PREPARATION
PERIOD OF UTERINE ENLARGEMENT
PERIOD OF UTERINE STRETCHING
GRAVID UTERUS:
NON GRAVID UTERUS, (UNILATERAL PREGNANCY):
a
b
c
d
e
f
g
h
31
1
6
8
9
12
16
20
22
24
28
32
DAYS OF PREGNANCY — RABBIT
PARTURITION

Fig. 1 Accommodation of the products of conception by the gravid uterus. Diagrammatic representation of the size and shape of the rabbit uterus about a fetus and its associated structures during pregnancy. Conceptus sites drawn to scale. Solid line indicates weight of gravid uterus less products of conception. See text for discussion.

curve from the period of uterine enlargement to the period of uterine stretching.

One of the principal observations of this study, and one which has meaning only when it is considered in relation to the other facts noted in this discussion, is that in any one rabbit the time required for transition from the spheroid to the cylindroid shape is relatively short. While this process occurs within the several days mentioned above, it appears to involve a much shorter period of time in any single instance. For example, in three rabbits out of sixteen studied thus far at these times the following special situation has been observed. Within a single uterine horn, two to four spheroidal conceptuses were found adjacent to one or more elongated (cylindroid) conceptus sites. The elongation in these instances involved the conceptuses lying in the cephalad, or tubal, end of the uterus. The cervix with its adjacent uterine tissue in each case has retained its characteristic form, and has not become ''effaced'' as it is soon after elongation of all conceptuses takes place in uteri containing several fetuses. A drawing of such a uterus, made from a photograph of a freshly fixed specimen, is shown in figure 2. In one case on the twenty-second day of pregnancy, while blood-circulation studies were being made upon a spheroidal conceptus, this spheroid merged with the next one during the course of the experiment. Most frequently on any one day, however, the shapes of all viable conceptuses were similar. For this reason, it is clear that when a spheroid conceptus attains a given maximum size, elongation occurs within a short space of time. Concurrently, the rate of uterine growth diminishes and the period of most rapid fetal growth begins.

One further observation with respect to the period of uterine stretching must be mentioned. It is that the maximum mesial and lateral diameters of the spheroid conceptus just prior to elongation are about the same as those found throughout the period of uterine stretching. It follows, therefore, that all accommodation by the uterus of the products of conception after the uterus ceases to grow is by enlargement in the longitudinal dimension. This holds true for the duplex uterus, if one may generalize from the situation in the rabbit. The extent to which this applies in the uterus bicornis and the uterus simplex has yet to be determined. The character of this enlargement is shown in figure 1, f, g, and h. This fact, which is so obvious when it is called to one's attention, has not been emphasized heretofore. Its meaning is suggested by the section which follows below.

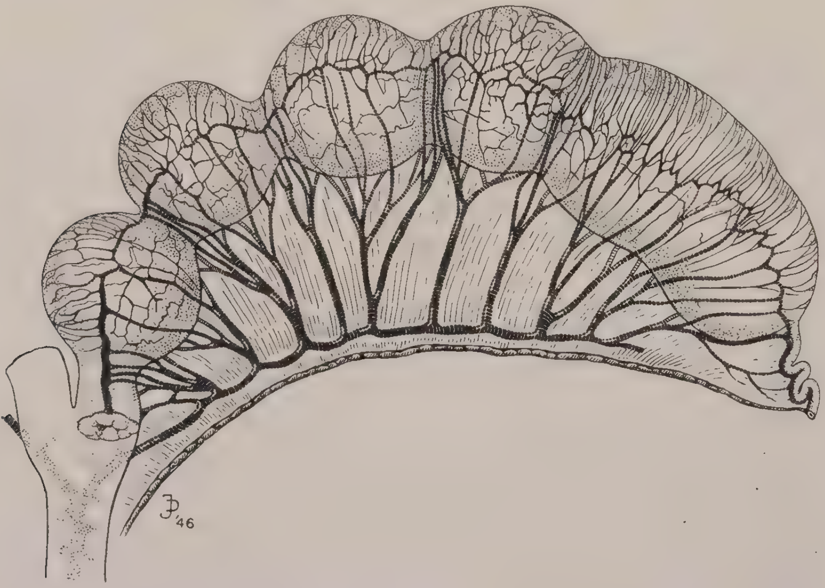


Fig. 2 Process of conversion from spheroidal to cylindrical shape in a gravid uterus. Drawing of a gravid uterine cornu of a rabbit on the twenty-second day of pregnancy. Four conceptuses are still spheroidal, two have elongated and merged to form a cylinder-like shape of the uterus. A third conceptus has nearly merged. Note rearrangement of venous network with change in shape. See text for discussion. The retrovaginal vein shown here is usually separated, and the arcuate vein in the broad ligament is bilaterally symmetrical.

II. FUNDAMENTAL RELATIONSHIPS

The physical relationships developing in the uterine wall as a result of growth and change in shape of the conceptus require explanation. For example, what factors contribute to the development of a cylinder which elongates during the period of very rapid fetal growth without a concommitant increase in diameter? The resistance to lengthening must be substantially less than it is to further increase in diameter. Reference to the general hydrostatic relationships involved show how, on physical grounds, this is possible. There is ample reason for making such an application since the pressure-volume-tension properties of hollow viscera have been shown to be comparable to those in simpler elastic systems (Sutherland, '09).

Physical conditions in the hollow elastic sphere. For simplicity, the spheroids of the second phase of accommodation of the products of conception (period of uterine enlargement) may be regarded as simple spheres. When conditions in actual situations are considered, allowances for the elliptical character of cross sections of the conceptus sites must be made, as in a previous study (Reynolds and Foster, '39).

In a hollow, elastic sphere, the hydrostatic relations are as follows:

$$T_s = \frac{r^2}{2} \cdot p$$

where T_s is the tension on the wall of an elastic sphere; r, the radius; p, the pressure. Figure 3a shows the direction of this force as a tangent of the surface at every point on the surface of the sphere.

It follows from this that in the uterus during the period of its enlargement to accommodate the products of conception, the tension will increase as the radius of the spheroidal conceptus increases, even though intra-uterine pressure may change but little.

Physical conditions in the hollow elastic cylinder. In this situation, two tension components will be found on the surface of the uterus. These are in accordance with the general considerations for such situations as shown by Frank and applied to conditions in the cardiovascular system (Hess, '28). These tension components (figure 3b, and c) are firstly, a circular tension component, and secondly, a longitudinal tension com-

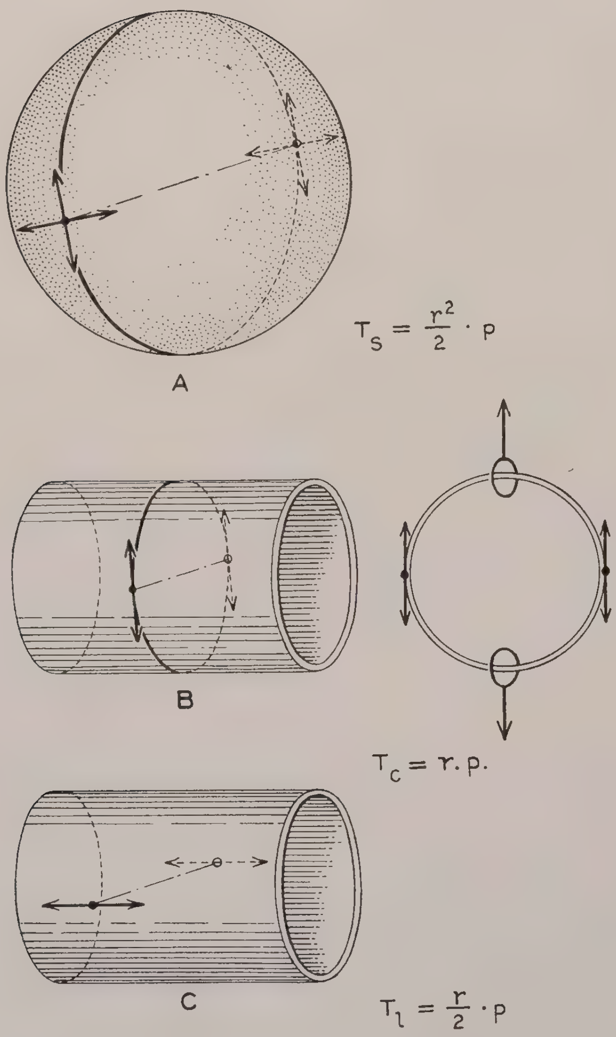


Fig. 3 Hydrostatic forces in hollow elastic bodies. Diagrams showing the direction of tensions on the surfaces of hollow elastic systems: A, sphere; B and C, cylinder. In the cylinder, the tension in a circular direction is just double the tension in a longitudinal direction. In the sphere, it is greater than either of the tension components in a cylinder having equal radius, pressure, and elastic properties. See text for discussion. If the elasticity is the same in each, and r and p are equal in the sphere and cylinder respectively, then $T_s:T_c:T_l::20:2:1$ (when $r = 10$ mm, and $p = 10$ mm H_2O).

ponent. These are in accordance with the following relationships:

$$T_c = r \cdot p \text{ and } T_1 = \frac{\pi r^2}{2\,\pi r} \cdot p = \frac{r}{2} \cdot p$$

where T_c is the circular tension component in an elastic cylinder, and T_1, the longitudinal tension component; r and p, as above.

The above relationships permit one to explain the fact that during the period of uterine stretching, accommodation of the products of conception is by increase in length alone, and not by increase in cross-sectional dimension. It is because the resistance of the uterus to lengthening is about half what it is to further increase in diameter. Experiments to test directly the applicability of this conclusion are necessary to establish this explanation as a fact. Until then it must be considered a plausible hypothesis.

III. FUNCTIONAL COROLLARIES OF THE HYDROSTATIC CHANGES IN THE UTERUS

Uterine growth. The role of tension as a stimulus for uterine growth has been discussed at length elsewhere (Reynolds, '37, '39) and need not be reviewed here. As noted above (fig. 1), marked uterine enlargement takes place only during the time when the uterus is spheroidal. This, as the relations noted in section II indicate, is a phase during which the tension at each point on the wall of the uterus about the conceptus is highest. Uterine growth virtually ceases as soon as the change in shape of the conceptus brings about a reduction in uterine tension. Thus tension on the uterus and growth of the gravid uterus are associated in a complementary manner in the accommodation of the products of conception, and both of these are determined by the shape and size of the products of conception.

Onset of labor. In the absence of experimental data, one can only speculate what the relations outlined in section II mean with respect to the end of gestation. A moment of consideration suggests, however, that labor may begin when the limit, or some other critical stage, of longitudinal stretching of the uterus is reached. As this time approaches the differential between circular and longitudinal tensions on the wall will be diminished in the manner described in an earlier paper (Reynolds and Foster, '39). There it was pointed out, ''that pregnancy in the rabbit comes to an end at the time when the maximal uterine tension becomes uniform over the surface of the whole organ. This appears to be, accordingly, the physical condition which signals impending parturition.'' This statement would appear to be fundamentally in agreement with the general relations noted in section II, above, although the existence of different circular and longitudinal tensions on the surface of the elongated uterus has not been recognized until now.

Hemodynamic conditions in the gravid uterus. This is the subject of other studies now in progress. It is pertinent to note at this time, however, that the gravid uterus presents circulatory conditions unique in the cardiovascular system. Blood in the maternal vascular system enters the uterine wall which is undergoing the progressive changes in the pattern of tension outlined in the previous section. These changes in tension must offer corresponding changes in the pattern of resistance to the entry of maternal blood into the uterus. Thus there will be higher resistance during the period of uterine enlargement (about the spheroid conceptus) than there is in the period of uterine stretching, at least until near term. Elongation of the uterus in the latter part of pregnancy must also bring about extensive re-orientation of the arrangement of the blood vessels within the uterus in a manner calculated to maintain efficient circulation of blood. This is a subject requiring special study.

Fetal growth. This follows in a very close manner the pattern of uterine tension. In the rabbit bearing a small litter the fetus on the sixteenth day of pregnancy weighs less than one one-hundredth of its weight 15 or 16 days later at birth. On the twentieth day, it weighs one twenty-fourth of its birth weight; and on the twenty-fourth day, it weighs about one-fifth of its weight at parturition (Hammond, '35). In grams, the weights involved are as follows:

Sixteenth day of pregnancy	0.58
Twentieth day of pregnancy	2.96
Twenty-fourth day of pregnancy	13.42
Birth (thirty-second day of pregnancy)	70.00

In the last week of pregnancy, therefore, at the time of lowered resistance in the uterine wall, a better flow of blood into the uterus is assured than it is earlier during the period of uterine enlargement. Moreover, the period of uterine elongation coincides with the period of most rapid transfer of substances across the placenta (Flexner and Pohl, '41) and with the most rapid increase in the daily increment of fetal weight (Hammond, '35).

To say that the pattern of tension in the uterine wall bears an important relation to fetal growth and shape, and vice versa, in no way lessens the importance of other structural and functional mechanisms which operate simultaneously in the fetus and its associated structures to provide for its growth and development. To enumerate and review these at this time lies beyond the scope of the present discussion.

Fetal welfare. From the foregoing discussion, it is clear that the proper integration of a number of structural and functional factors governs the relations of growth of the fetus to its environment within the uterus. For example, tension on the uterine wall attains a maximum value at the time of maximum spheroid size of the uterus (excluding from discussion the special situation that prevails at term). This is so because the radius is maximal. If the premise is true that blood flow through the maternal vessels in the uterus is affected by uterine wall tension (observations supporting this point have been made and will be reported separately) it follows that transient physiological ischemia will develop at this time, and that it will be relieved as soon as elongation takes place. It is interesting to note, therefore, at what period in late pregnancy fetal death begins to be encountered. Counts of resorbing fetuses in gravid uteri are summarized in table 1.

Reference to this table shows that fetal death in late pregnancy, as evidenced by resorption, has been observed only on and after the twentieth day of pregnancy in the rabbit. These observations show that fetal death most commonly begins to be encountered near the end of the period of uterine enlargement when tension on the spheroidal conceptus is at a maximum. Once elongation has taken place, viability until near term seems relatively assured. The inference is clear, therefore, that conditions critical for the welfare of the fetus develop as the spheroid conceptus attains maximum size.

TABLE 1

Resorbing fetuses observed in unilateral and bilateral pregnancies related to the stage of pregnancy.

DAY OF PREGNANCY	NUMBER OF LITTERS	NUMBER OF LIVING FETUSES	NUMBER OF RESORBING FETUSES	PER CENT RESORBING FETUSES
12	7	48	0	0
16	7	69	1	1.3
18	1	8	0	0
20	6	49	3	5.8
22	7	59	4	6.2
24	6	38	6	13.4
28	8	49	6	10.9
30	1	4	1	20.0

While the practical significance of this conclusion is clear with respect to fetal welfare, the physiological basis of abnormal hydrostatic relationships within the uterus merits extended study.

SUMMARY

1. The uterus may be said to accommodate the products of conception by a succession of stages which include a phase of preparation (mitotic proliferation of uterine elements); uterine enlargement (hypertrophy of all uterine elements); and uterine stretching (cessation of uterine growth with rapid enlargement of the products of conception).

2. The period of uterine enlargement is characterized by the presence of conceptuses that are spheroidal in shape. These

increase to a maximum size. As the conceptus elongates, uterine growth (enlargement) ceases and stretching begins. From this point until term, in the rabbit, accommodation of the products of conception is by elongation of the uterus and its contents without further increase in diameter. The conversion process of elongation of spheroids requires a short period of time.

3. Theoretical considerations offer a basis for explaining the foregoing observations. In hollow elastic spheres, tension on the surface of the sphere is high ($T_s = \frac{r^2}{2} \cdot p$). With elongation tension in a circular direction becomes less than in a sphere of equal diameter ($T_c = r \cdot p$) and tension along the longitudinal axis is still less ($T_l = \frac{r}{2} \cdot p$).

4. Accommodation of the conceptus during the period of uterine stretching by elongation occurs because resistance to lengthening is about one-half what it is to further increase in diameter.

5. Functional corollaries of the above general relationships are discussed. Uterine growth in the period of uterine enlargement is directly related to tension on the uterine wall. Circulation of maternal blood through the uterus bears an inverse relationship to the tension on the wall of the uterus; fetal growth, which is very small until the period of uterine stretching commences (i.e., a time of lower tension on the uterus) is inversely related to the tension on the uterine wall. Fetal death (resorption) is observed to occur most frequently at the end of the phase of uterine enlargement (maximum spheroid size) when tension on the wall of the uterus is maximal just prior to elongation.

LITERATURE CITED

Ancel, P., and P. Bouin 1910 Recherches sur les functions du corps jaune gestatif. I. Sur déterminisme de la préparation de l'utérus a la fixation de l'oeuf. J. de Physiol. et de pathol. gén., vol. 12, p. 1.

Corner, G. W., and W. M. Allen 1929 Physiology of the corpus luteum. II. Production of a special uterine reaction (progestational proliferation) by extracts of the corpus luteum. Am. J. Physiol., vol. 88, p. 326.

Flexner, L. B., and H. A. Pohl 1941 The transfer of radioactive sodium across the placenta of the rabbit. Am. J. Physiol., vol. 134, p. 344.

HAMMOND, J. 1927 The Physiology of Reproduction in the Cow. Cambridge, Cambridge Univ. Press.

——— 1935 The changes in the reproductive organs of the rabbit during pregnancy. Trans. on the Dynamics of Development, vol. 10, p. 93 (Russian) (see Reynolds, '39).

HESS, W. R. 1928 Die Gesetze der Hydrostatik und Hydrodynamik. In Bethes' Handbuch der Normalen und Pathologischen Physiologie, VII, vol. 2, p. 889.

IVY, A. C. 1942 The functional anatomy of labor, with special reference to the human being. Am. J. Obst. Gynecol., vol. 44, p. 952.

REYNOLDS, S. R. M. 1937 Hormonic and physical factors in uterine growth. Symposia on Quant. Biol., Cold Spring Harbor, L. I., N. Y., vol. 5, p. 84.

——— 1939 Physiology of the Uterus. New York and London, Paul B. Hoeber.

REYNOLDS, S. R. M., AND F. I. FOSTER 1939 Physical conditions in the uterus governing the duration of pregnancy. Anat. Rec., vol. 75, p. 175.

SIEGMUND, H. 1930 Über die Abhängigkeit der Uterusmuskulatur von den Funktionsphasen des Ovariums. II. Die Reaktionslage der Uterusmuskulatur während der Gravidität und im Puerperium bei der Ratte. Arch. f. Gynäkol., vol. 140, p. 583.

SUTHERLAND, W. 1909 Note on the paper by W. Osborne, entitled ''The elasticity of rubber balloons and hollow viscera.'' Proc. Roy. Soc., s. B, vol. 81, p. 497.

A SILVER-GELATIN METHOD FOR STAINING NERVE FIBERS[1]

ANTHONY A. PEARSON AND SARAH LEA O'NEILL
Department of Anatomy, Baylor University College of Medecine, Houston, Texas

ONE PLATE (SIX FIGURES)

There is still a search for better methods to demonstrate the finer anatomy of the nervous system. While there are a number of good methods, many of them are unsatisfactory for embryos and fail to stain nerve fibers in tissues which have required some decalcification. This is a modification of the Warthin-Starry stain for Treponema pallidum, as outlined by Farrier and Warthin ('30) and Galantha ('32). An abstract of this method has been recently published (Pearson and O'Neill, '46). The more important steps of the method will be briefly outlined.

Good fixation is essential. The usual fixatives such as 10% formalin, formol-acetic-alcohol, and Bouin's fluid give good results. Perfusion of the fixative is recommended when possible. Extraction in 70% alcohol over a period of several weeks is thought to improve the staining quality of the tissue. During the extraction the alcohol should be changed frequently. Bodian ('37) has discussed more completely the role of a number of fixatives in staining nervous tissue with silver stains.

Bony tissues may be decalcified in either 2 to 5% nitric acid or 5% trichloracetic acid in 70% alcohol. The acid alcohol should be changed daily until the decalcification is complete.

---

[1] This research was aided by the fund contributed by the M. D. Anderson Foundation.

As the tissue is dehydrated after decalcification, several changes in 1 to 2% ammoniated alcohol is recommended. The tissue should then be cleared, embedded, cut, and the paraffin removed in the usual manner. The sections now ready to be stained are placed in adjusted water.

It is believed that the uncertainty of some of the silver methods is due largely to variations in the hydrogen-ion concentration. Farrier and Warthin ('30) found that the quality of the stain increased as the pH values approach 4.4 which they considered to be optimum. They recommend that citric acid be used to adjust the water on the acid side since it does not react with either silver nitrate or hydroquinone.

Water adjusted to approximately a pH of 4.4 can be prepared by adding 15 drops of 1% citric acid to a liter of distilled water. We have found this to be quite satisfactory. This adjusted water should be used for all solutions until the sections are ready to be toned in gold chloride.

The slides should be placed in 2% silver nitrate at 60°C. for 45 minutes. Mix rapidly 2 cm^3 of 1% hydroquinone, 5 cm^3 of 2% silver nitrate and 20 cm^3 of 3% gelatin. All of these should be at about 60°C. Place the slides in this solution and keep the temperature roughly at 60°C. Agitate gently until the sections turn a golden brown. Wash in adjusted water. Distilled water is used from here on.

The sections are ready to be toned in a solution of 1% gold chloride(acid yellow) for 5 to 10 minutes. They are then washed in distilled water and placed in 1–2% oxalic acid until the sections turn a light purple color, which is usually about 1–2 minutes. The sections are washed again in distilled water. The residual silver may be removed in a solution of 5% sodium thiosulphate (5–10 minutes). Wash the slides in distilled water, dehydrate, clear, and mount in balsam. All glassware must be kept chemically clean.

The above method has proved very satisfactory in staining human and mouse embryos and fetuses. It makes possible the staining of ten slides in a glass basket at one time with uniform results. Nerve fibers to regions such as the sensory epithelium of the membranous labyrinth (fig. 1) and the autonomic plexuses within the wall of the fetal gut were sharply defined. This method will stain red blood cells (fig. 2) and may prove to be useful in studying the pattern of the blood stream. More research will be necessary to determine the possibilites of this method.

The figures (1–6) illustrating this stain are photomicrographs of selected sections of a 15-day-old mouse fetus fixed in Bouin's fluid.

LITERATURE CITED

Bodian, D. 1937 The staining of paraffin sections of nervous tissues with activated protargol. The role of fixatives. Anat. Rec., vol. 69, pp. 153–162.

Farrier, R., and A. S. Warthin 1930 A study of the effect of pH upon the third improved Warthin-Starry method for demonstrating Spirocheta pallida in single sections. Am. J. Syph., vol. 14, pp. 394–401.

Galantha, E. de 1932 Modified silver stain for Treponema pallidum. Am. J. Clin. Path., vol. 2, p. 63.

Pearson, A. A., and Sarah L. O'Neill 1946 A modified silver stain for nerve fibers. Anat. Rec., vol. 94, pp. 545–546.

PLATE 1

EXPLANATION OF FIGURES

1 Nerve bundle to a crista of a semicircular canal of a fetal mouse.

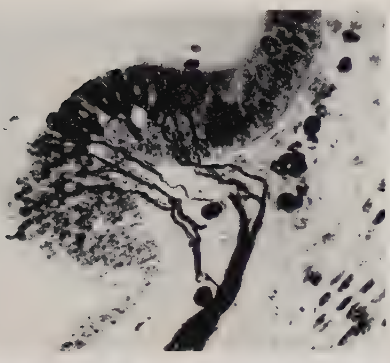
2 A bipolar nerve cell on the spinal portion of the spinal accessory nerve in a fetal mouse.

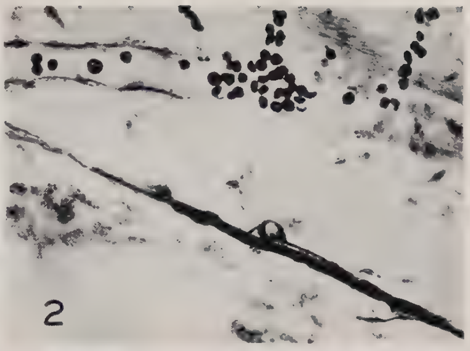
3 Ganglion cells of the vagus nerve in the fetal mouse.

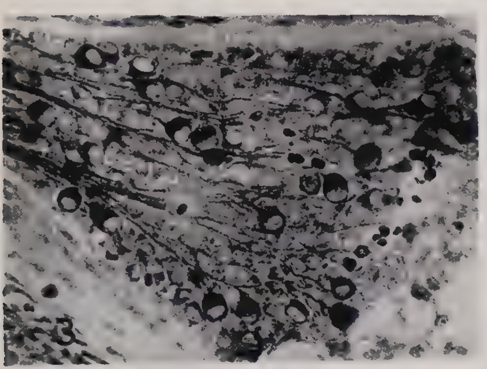
4 Nerve plexus in the wall of the esophagus of a fetal mouse.

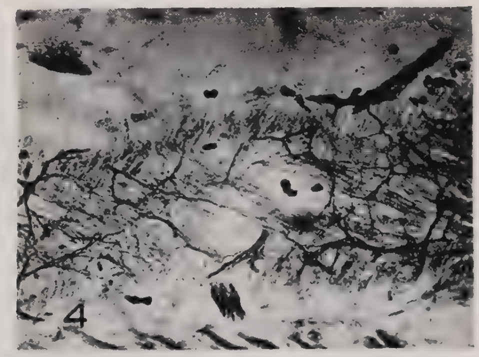
5 A cross section of the medulla oblongata of a fetal mouse.

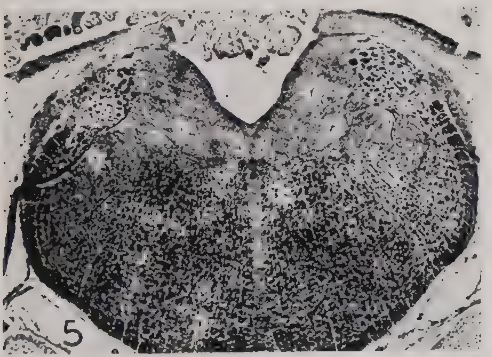
6 A cross section of the spinal cord at the level of the first cervical nerve of a fetal mouse.

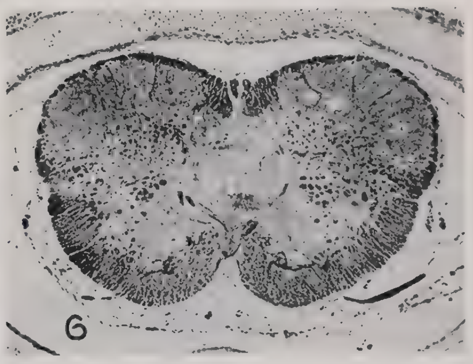

1


2


3


4


5


6

GLASS AND STEEL CONTAINERS FOR HUMAN ANATOMY

J. M. ESSENBERG

Department of Anatomy, The Chicago Medical School and Loyola University School of Medicine, Chicago, Illinois

TWO FIGURES

For the preservation and mounting of large preparations such as hermaphrodites, situs inversus viscerum, non-rotation of the gut, special dissections of the body, etc., large containers are essential. Before World War II, glass jars large enough to accomodate the torso of the human body were imported from Germany. These, however, proved unsatisfactory for permanent mounts. Because of accidents and of sudden fluctuations of the temperature, breakage resulted with serious consequences. After a few accidents of this kind, the writer attempted to produce a container of more durable structure. The result is illustrated in figure 1, which has stood the test of 10 years with neither breakage nor leakage.

This container, housing the entire body of a hermaphrodite, is, in outside dimensions, 70 inches in height, 21 inches in width and 12 inches in depth. The rectangular frame is made of 3-inch angle-iron, $\frac{1}{4}$ inch in thickness. All of the joints of the angle-iron case are machined after the fashion of a picture frame, i.e., 45° angle, welded and filed smooth, especially the exposed surfaces. One end of the finished frame is welded or riveted to a rectangular steel base, $\frac{1}{2}$ inch in thickness, 27 inches in length and 18 inches in width. The welding of these parts is best done on the inside of the frame. To each corner of the base is attached a 3-inch, swivelled trucking caster which facilitates the moving of the container from place to place.

Plate glass is the best for this purpose. Sheet glass, however, is less expensive and serves the purpose almost as well. The thickness of the glass should be ½ inch and free of imperfections. The length and width of the plates should be at least ¼ inch less than the available space. This allowance is given for cement and expansion. The side plates should be cemented in place first, then the end plates and finally the floor plate is installed, figure 2.

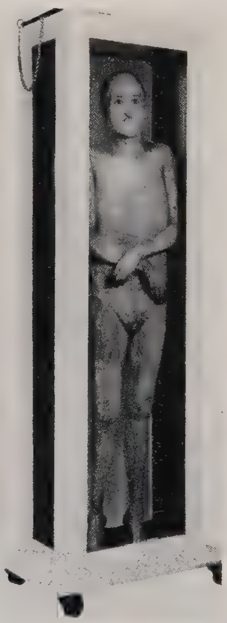


Fig. 1 Glass and steel container completed.

Any aquarium cement of good quality is satisfactory for this purpose. It is put on the angle-iron, more or less evenly distributed and then the glass plate is placed on it and slight pressure applied until a complete seal is made. Between the steel and the glass the layer of cement can be about ¼ inch in thickness; between glass and glass it can be less than that but not less than $\frac{1}{8}$ inch and well packed. It is important that at no place glass rests on metal or on glass without sufficient cement between these surfaces.

When the glass plates are installed in the steel frame, the inside surfaces are cleaned, the specimen mounted and the container filled with fluid. It will be noted that the fluid contacts only glass and cement but no metal.

If it becomes necessary to change the fluid of the container, siphoning will serve the purpose. If, however, it is desired to have a direct outlet, it can be done as follows: A large sized stopcock is attached to the metal basal plate by screw, a hole is drilled (prior to installment) in the glass floor plate and the two, stopcock and glass floor plate, connected by a glass tube, are sealed with cement.

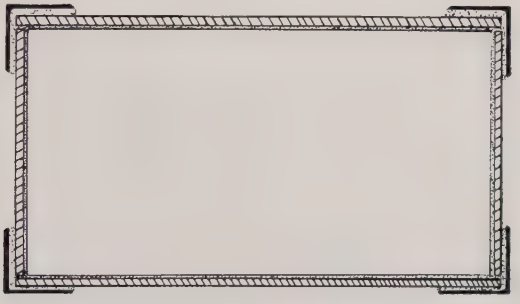


Fig. 2 Transverse section of container in region of floor plate. Angle-iron in solid black, glass in oblique lines, cement in stipple.

The strength of the frame and the glass is undoubtedly more than required for this size of a container and reductions can be made if desired. However, considering the amount of fluid in it and also the fact that it may have to be moved about, over-strength is an advantage.

It is apparent that a container of this kind can be constructed by any mechanic who has the equipment for welding steel. The expense is comparatively little especially if sheet glass is used instead of plate glass. Any size or shape can be made to suit the purpose. The outside surfaces should be finished neatly and, to give it a good appearance, enameled or chrome-plated.

FUSION OF NOTOCHORD TO NEURAL TUBE IN A HUMAN EMBRYO OF THE SIXTH WEEK

BRADLEY M. PATTEN
University of Michigan Medical School, Ann Arbor

ONE PLATE (THREE FIGURES)

There is a very proper reluctance about publishing papers based upon single instances of abnormal development. Embryological malformations are far from simple phenomena, and one naturally attempts to find a number of cases to form the basis of a report, since only a group of related defects, or a series of individuals showing different stages in the genesis of a given defect, are likely to help much in understanding the processes involved. Consequently, when an embryo showing a local fusion of the notochord with the developing spinal cord came into our Michigan collection, it was not reported for several years in the hope that other such cases might be encountered. Fusions of the notochord at its cephalic end with Rathke's pocket or with pharyngeal epithelium are not uncommon occurrences. Fusion with the neural tube at cord levels is apparently most unusual. No embryo with this condition has been seen in the interval, nor has a search of the literature brought to light any published description of such a case. When, finally, questioning of a group of embryologists of wide experience revealed that none of them had seen any case of the type, it seemed desirable to publish photographs of the condition. If such a fusion of the notochord with the neural tube is unique, it should be on record; if, as is more probable, similar cases have been seen but not reported, the reporting of this case may bring them out of seclusion, and lead toward the accumulation of sufficient data for intensive study.

The embryo in question was sent in to this laboratory without any accompanying data. The specimen, measuring 11.2 mm in crown-rump length, had obviously been promptly fixed and appeared to be in excellent condition so it was catalogued (EH 268) and designated for serial sectioning. As luck would have it, we already had good transverse series in this age range, so this embryo was cut sagittally, which proved to be the ideal orientation to show the notochordal fusion to best advantage. Plate 1, A, shows, for general orientation, an entire section from near the middle of the series. The rectangle indicates the location of the moderately magnified photomicrograph appearing as B in the same plate. This photograph shows, between the mesenchymal clusters constituting the primordia of the centra of the seventh cervical and the first thoracic vertebrae, an abrupt deviation of the notochord. Here the notochord executes a U-shaped bend dorsad, to meet and fuse with the neural tube. At the point of fusion the floor plate of the neural tube is pulled toward the notochord. The entire picture suggests the two structures had fused at a considerably younger stage when they lay close together, and that the persistence of this fusion as the two structures became separated pulled both of them out of their normal alignment. The high power photomicrograph (C) shows, at the point of union, closely packed notochordal cells adherent to the marginal layer of the spinal cord. The connection involves four 10 μ sections.

As to the time and place of the origin of this condition, one can perhaps formulate a reasonable hypothesis. Allowing for the four occipital somites, the interspace between the seventh cervical and the first thoracic vertebra, where the adhesion appears, is opposite the eleventh somite. It seems probable that the adhesion was formed at about the 10- or 11-somite stage when the cells destined to form the floor of the neural tube and the cells giving rise to the notochord were juxtaposed in the growth center just cephalic to the primitive streak. Judging from the very small extent of the adherence, whatever induced it must have been operative for only a few hours since at this age pairs of somites are being cut off at the rate of two to three a day, and this defect is well within the level of a single pair of somites. With the growth and differentiation which has intervened since its formation, the area of adhesion has come to lie relatively much farther cephalad in the body and the portion of the notochord subsequently formed appears entirely normal in its configuration and relations.

It is very difficult to guess just what the resultant abnormalities at later stages would have been had this embryo been permitted to continue its development. One can surmise that the intervertebral disc might have been weakened and prone to rupture where it developed around the misplaced notochord. It is conceivable that the aberrant notochordal tissue might have become involved in the formation of some sort of a tumor-like mass or cyst. The disturbance of the marginal layer of the cord scarcely seems sufficient to interfere with the development of the funiculi adjacent to the ventral sulcus, although this would have to be considered as a possibility. Beyond such rather futile speculations one cannot go in the absence of evidence such as would be furnished by other embryos showing similar conditions at different ages.

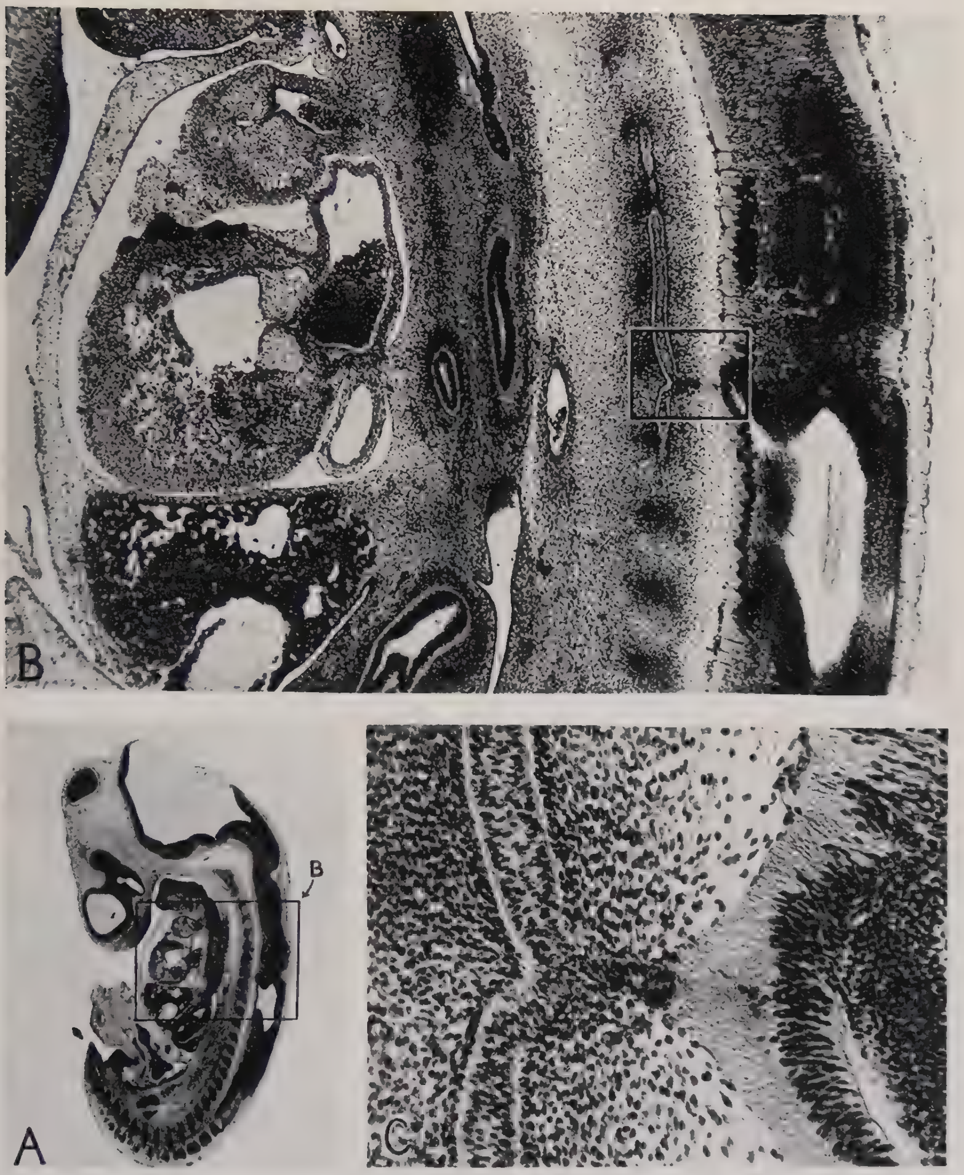
PLATE 1

EXPLANATION OF FIGURES

A Photomicrograph of entire section near middle of series with rectangle indicating the area shown more highly magnified in B.

B Photomicrograph at moderate magnification to show the topography in the neighborhood of the adhesion. The rectangle indicates the area shown in C in higher magnification.

C Photomicrograph (× 200) showing the details in the area of fusion.


B
B
A
C

RESPONSE OF PREPUBERAL MALE MICE TO EQUINE GONADOTROPIN

D. H. BISHOP AND J. H. LEATHEM
Department of Zoology, Rutgers University, New Brunswick, New Jersey

Many factors may influence the response of the gonads to gonadotropic substances and it was very quickly recognized that the age of the animal was one of these factors (Smith, '26; Smith and Engle, '27). Nevertheless a clear understanding of the relationship of age to reactivity has not been gained because, as Price and Ortiz ('44) point out, hormone dosage, periods of treatment and methods of estimating results have all been subject to variation. The reactivity of the rat to equine gonadotropin (pregnant mare serum) at ages ranging from 1 to 50 days has been studied by Price and Ortiz ('44). These investigators found that the age at which an organ responds maximally will vary with different organs but that some response is evident in male rats killed as early as day 6. Since equine gonadotropin is a potent gonadotropic material and to our knowledge has not been carefully studied in the mouse, we have undertaken a study of the response of the prepuberal male mouse to this hormone to estimate the time of maximal organ responses.

MATERIALS AND METHODS

Male mice of 5, 10, 15 and 20 days of age were used. The mice were the "Swiss albino" strain raised in our laboratory. Equine gonadotropin (Gonadogen, Upjohn[1]) was administered as a single subcutaneous injection of 50 I.U. (0.1 cm^3).

---

[1] Equine gonadotropin, Gonadogen, was provided through the kindness of Doctors G. F. Cartland and M. H. Kuizenga of the Upjohn Co., Kalamazoo, Mich.

The single injection permitted a minimum of disturbance to the animals which were all kept with the nursing mothers until autopsied. The injected mice were autopsied with their littermate controls 120 hours after the injection and the fresh weight of the testis, seminal vesicles, spleen and kidney was recorded. The testes and adrenals were fixed in Bouin's fluid, sectioned and stained with Harris hematoxylin and eosin for histological examination.

RESULTS

Autopsy at 5 days of age

A group of mice were killed at 5 days of age to determine organ weights at this age and these data are presented in table 1.

TABLE 1

Response of prepuberal male mice to 50 I.U. of equine gonadotropin (PMS)

TREATMENT	AGE AT AUTOPSY	NO. OF MICE	AVERAGE BODY WT.	AVE. WT. OF ORGANS	
				Testes	Sem. ves.
			gm.	*mg.*	*mg.*
none	5	9	3.8	6.4	1.2
none	10	6	5.1	12.9	1.5
PMS	10	10	4.8	16.2	4.8
none	15	6	7.3	27.0	2.0
PMS	15	9	7.1	27.8	10.6
none	20	6	10.3	58.7	3.4
PMS	20	9	11.1	59.8	18.0
none	25	5	13.4	85.9	4.4
PMS	25	8	13.2	79.8	15.1

Autopsy at 10 days of age

Mice were injected with equine gonadotropin at 5 days of age and autopsied with their littermate controls when all were 10 days of age. The hormone did not significantly alter the weight of the spleen or kidney nor did it impair body weight increase. Testis weight was 25% greater in the injected mice than in the controls but the increase was not significant.

The normal increase in seminal vesicle weight which might be anticipated between the fifth and tenth postnatal days was slight and not significant. Injection of equine gonadotropin, however, had a definite stimulating action at this age increasing seminal vesicle weight 220% (table 1).

Histological evidence of hormone stimulation was observed in the testis as indicated by the definite increase in interstitial tissue. This increase was quite apparent since the interstitial tissue in control animals consisted of relatively sparse and widely scattered cells. Spermatogenesis was not accelerated by hormone administration. The adrenal was also examined since it is well known that the X-zone in the mouse is very sensitive to androgens, these hormones causing its rapid disappearance. A narrow X-zone, however, was observed in the adrenal cortex of both the experimental and control mice. Although it is apparent that androgens are being released by the testis as evidenced by seminal vesicle weight increase either the quantity is not sufficient to cause X-zone disappearance or the X-zone is not influenced at this stage of development.

Autopsy at 15 days of age

The mice injected at 10 days of age were autopsied with their littermate controls when 15 days old. Experimental results essentially simulated those observed in the younger age group in that weight of the testis, spleen and kidney was not changed and body weight increased normally. The seminal vesicles responded markedly in this group being 430% heavier than non-injected controls of the same age (table 1). Between the fifth and fifteenth days of age the seminal vesicles increased 60% normally whereas equine gonadotropin caused these organs to be 700% heavier than those from the 5-day-old animal.

Histologically an increase in interstitial tissue was observed in the testes in the absence of a stimulating effect on the seminiferous tubules. The adrenal X-zone too, provided evidence for a hormone effect since it had disappeared in experimental mice but was quite definite in control mice.

Autopsy at 20 days of age

The mice injected when 15 days old were autopsied when 20 days old. Testis, spleen and kidney weight was not altered and a normal increase in body weight was recorded. Seminal vesicle weight increased 430% following the administration of equine gonadotropin in comparison with littermate controls. This degree of response was the same as that seen with mice killed at 15 days of age. Seminal vesicle weight increased 200% normally from the fifth to twentieth days of age whereas equine gonadotropin induced a 1400% organ weight increase during the same period.

Spermatogenic activity was not hastened by hormone administration but interstitial tissue was definitely increased. An increase in interstitial tissue was seen in normal mice at this age in comparison with younger animals but the hormone action was more pronounced.

The adrenal X-zone was present in all control mice but had disappeared completely in the hormonally treated mice.

Autopsy at 25 days of age

Mice injected when 20 days old were killed on day 25 at which time weight of the testis, spleen and kidney was unchanged in comparison with controls of the same age. Equine gonadotropin increased seminal vesicle weight 240% above that of littermate controls but this response was less than that observed in the 15- and 20-day-old groups (table 1). Seminal vesicle weight normally increased 250% between the fifth and twenty-fifth days of age whereas an 1100% weight increase was induced by hormone administration during the last 5 days.

Spermatogenesis was not accelerated but interstitial tissue was increased in these animals. Androgen secretion was sufficient to cause a disappearance of the adrenal X-zone invariably observed in littermate control mice.

DISCUSSION

The administration of equine gonadotropin to prepuberal male mice reveals their ability to respond to hormone treatment. This response consists of an increase in the interstitial tissue of the testis and a consequent increase in seminal vesicle weight. Normally, the seminal vesicles enlarge, at least slightly, at each 5-day interval between 5 and 25 days of age. There appears to be a somewhat greater increase in seminal vesicle weight between 15 and 20 days of age (at autopsy) than at other intervals and this is correlated with an apparent increase in testicular interstitial tissue. When hormone is administered, a marked increase in seminal vesicle weight is observed in all age groups with a maximal effect observed in mice 15 or 20 days old at autopsy. The stimulating action of equine gonadotropin on the accessory reproductive organs of the prepuberal mouse was observed by Starkey and Leathem ('39) but only one age group was studied. Price and Ortiz ('44) observed maximal stimulation of seminal vesicles in rats injected from 20 to 26 days of age or a somewhat later time than found for the mouse in spite of a larger total dose being injected.

Following the normal growth of the testis it was observed that testis weight doubled at each 5-day interval except for those killed at 25 days of age. Marvin and Meyer ('43) found testis growth rate to increase after the eleventh day in the rat. Equine gonadotropin failed to increase testis weight above that of littermate controls and did not accelerate spermatogenesis. Interstitial tissue, however, was invariably increased by the 50 I.U. unit dosage. Similar results were obtained in 12-day-old mice injected with a total of 100 I.U. over a 10-day period (Starkey and Leathem, '39). Unlike the mouse, however, the testis of the prepuberal rat readily responds to equine gonadotropin with an increase in weight which Price and Ortiz ('44) found to be maximal for rats killed at 14 days of age. Extensive treatment of the rat with equine gonadotropin had previously been shown to be capable of increasing testis weight but in no instance was spermatogenesis hastened (Cole, '36; Moore, '36).

The differences between the prepuberal mouse and rat as to time of maximal seminal vesicle weight response and the failure of the mouse to show an increase in testis weight may be due to experimental differences. In our experiments, equine gonadotropin was given as a single subcutaneous injection of 50 I.U. and autopsy performed 120 hours later whereas Price and Ortiz ('44) injected 10 r.u. (20 I.U.?) daily for 6 days and killed their animals the following day. However, the manner in which the hormone was given will not explain differences in results since equine gonadotropin is equally effective in male rats when given as a single or as divided subcutaneous injections (Leathem, '44). Therefore, when seminal vesicle weight is the determining factor, a state of maximal responsiveness is reached earlier in the mouse than in the rat whereas when testis weight increase is used the mouse, unlike the rat, fails to show a weight response. Starkey and Leathem ('39) did not increase testis weight in the prepuberal mouse with a 100 I.U. dosage.

The adrenal X-zone will promptly disappear when testosterone propionate is administered (Starkey and Schmidt, '38) and equine gonadotropin will indirectly cause X-zone disappearance (McPhail and Read, '42). In contrast, a survey of the literature indicates that estrogens will not influence the X-zone (Leathem and Silverman, '45). Examination of the adrenals of the prepuberal mice treated with equine gonadotropin would then indicate the release of androgen by the stimulated testis. An X-zone was found in the adrenals in all control mice whereas this zone had disappeared in all experimental mice except the group killed at 10 days of age. The adrenal X-zone in the 10-day-old mouse is a very narrow band of closely packed and darkly staining cells and was either nonresponsive to androgen and/or the amount of androgen released was insufficient.

SUMMARY

Equine gonadotropin was injected into mice when 5, 10, 15 and 20 days of age. Five days later the mice were studied for organ responses and were found to respond by an increase in interstitial tissue of the testis, an increase in seminal vesicle weight and, in most cases, a disappearance of the adrenal X-zone. Testis weight was not increased and no apparent hastening of spermatogenesis was observed. No effect on the weight of the kidney or spleen was recorded and body weight gains were normal.

Seminal vesicle weight was increased in all mice receiving the hormone, being at least 220% greater than the controls. Stimulation was maximal in mice killed at 15 and 20 days of age. Sufficient androgen was secreted by mice 15–25 days old at autopsy to cause a disappearance of the adrenal X-zone invariably observed in control mice.

LITERATURE CITED

Cole, H. H. 1936 On the biological properties of mare gonadotropic hormone. Am. J. Anat., vol. 59, pp. 299–331.

Leathem, J. H. 1944 Action of equine gonadotropin in normal and hypophysectomized immature male rats. Am. J. Physiol., vol. 140, pp. 561–565.

Leathem, J. H., and S. Silverman 1945 Action of stilbestrol on the female mouse adrenal gland. Proc. Penna. Acad. Sc., vol. 19, pp. 60–65.

Marvin, H. N., and R. K. Meyer 1943 The effects of antigonadotropic serum on the early postnatal development of the reproductive system of the male rat. Anat. Rec., vol. 85, pp. 177–194.

Moore, C. R. 1936 Response of immature rat testes to gonadotropic agents. Am. J. Anat., vol. 59, pp. 63–88.

Price, D., and E. Ortiz 1944 Relation of age to reactivity in the reproductive system of the rat. Endocrinology, vol. 34, pp. 215–239.

Smith, P. E. 1926 Hastening development of female genital system by daily homoplastic pituitary transplants. Proc. Soc. Exper. Biol. and Med., vol. 24, pp. 131–132.

Smith, P. E., and E. T. Engle 1927 Experimental evidence regarding the role of the anterior pituitary in the development and regulation of the genital system. Am. J. Anat., vol. 40, pp. 159–217.

Starkey, W. F., and J. H. Leathem 1939 Effect of mare serum on the male and female mouse. Am. J. Physiol., vol. 127, pp. 751–754.

Starkey, W. F., and E. C. H. Schmidt 1938 The effect of testosterone propionate on the X-zone of the mouse adrenal. Endocrinology, vol. 23, pp. 339–344.

FORM AND BEHAVIOR OF ADULT MAMMALIAN SKELETAL MUSCLE IN VITRO

IRENE A. POGOGEFF AND MARGARET R. MURRAY
Surgical Pathology Laboratory of the Columbia University College of Physicians and Surgeons and the Department of Surgery of the Presbyterian Hospital, New York

THREE PLATES (SEVENTEEN FIGURES)

Since the middle of the nineteenth century, regeneration in the skeletal muscle of adult mammals has been studied in tissue sections and described by numerous workers. Forbus ('26), describing regeneration in the rectus abdominis after pneumonia, gives a comprehensive review of the literature dealing with the subject up to that time. More recently, Pappenheimer ('39) has noted in the rat practically complete restoration to normal structure on the part of skeletal muscle tissue degenerated as a result of vitamin E deficiency; and Favata ('44) has reported complete regeneration in dog muscles after experimentally produced gas gangrene.

Many workers, among them Levi ('16), Lewis ('15), Freidheim ('31), de Renyi and Hogue ('31), Burkhardt ('36), Chèvremont ('40), Mauer ('39), and Szepsenwohl ('42), have employed embryonic skeletal muscle as the subject of studies in vitro. In this material the early stages of myogenesis from potential muscle tissue may be followed, but it has been doubted that this process in the embryo is entirely similar to the course of regeneration as it takes place in injured muscle which is fully differentiated.

Normal adult skeletal muscle can readily be grown in tissue culture (Pogogeff and Murray, '45), the morphology and physiology of the cells studied under high dry magnification, and its regenerative behavior observed directly. These observations are reported because of the present lack of agreement as to the morphology of regeneration in skeletal muscle, as to the functional significance of the characteristic fiber structures, and as to the relationship of innervation to growth and function.

The material employed in this study was skeletal muscle from the region of the knee-joint and thigh of normal adult rats, and human skeletal muscle obtained chiefly from amputations and radical mastectomies. The muscle was cultivated by the Maximow method, in which the culture remains undisturbed for long periods of time on the same coverslip, where it is washed and fed. The tissues were explanted and grown in a medium consisting of human placental serum, chick embryo extract, chicken plasma, Simms' ultrafiltrate and Simms' buffered saline solution (Simms and Sanders, '42). Occasionally rat serum or plasma was used in some of the rat cultures, but this seemed to effect no improvement in their growth. The cultures were routinely washed and fed at least three times a week. Approximately ninety rat cultures and eighty human cultures were studied. In some of the human cultures, attempts were made to record the history of individual cells by photographing the cell daily or at intervals of a few days throughout its life.

Skeletal muscle of adult humans and rats showed similar growth patterns in vitro except that the rat tended to form longer muscle ribbons (figs. 10, 11, 15). A human fiber of 200 μ was regarded as fairly long; but rat fibers sometimes grew to a length of 1600 μ, with a width of 8–9 μ.

The cultures of adult muscle are generally similar in appearance to those of embryonic muscle except that in the latter the cells have on an average a greater number of nuclei, and the fibers may be broader at times.

The average time elapsing before the appearance of clearly recognizable muscle-cells in the adult rat cultures was about 13 days, and in the human 11½ days. Since fibroblasts and other elements of granulation tissue make their appearance much earlier than this in both tissue cultures and granulating wounds, it seems probable that this long lag-period shown by skeletal muscle is a factor in the generally poor end-result of muscle-regeneration in vivo following trauma. By the end of a fortnight after a wound is made, cicatricial tissue has usually made a good start, and in many conditions is well-organized, thus blocking mechanically, if in no other way, the outgrowth of the new-forming muscle-buds. There is considerable individual variation, however, in the length of the lag-period for muscle-cells; in the rat the range is from 6 to 19 days, and in the human from 8 to 17 days in vitro.

Regenerating cell-forms. The emigrating muscle-cells were found as isolated spindle-cells, as fragments with round, rectangular or club-shapes, or as ribbon-like forms budding out from within the explant (figs. 1–5, 9–11). In the case of the human cultures, these ribbon-like structures did not attain the length that they did in the rat, nor did they tend to remain attached to the explant. They seemed rather to wander out in the clot and exist as independent cells. In the rat cultures, especially in the earlier days of culture, one found large, multinucleate, macrophage-like forms. Broad, flattened, multinucleate cells were to be seen in the early days of culture of the human muscle, but these did not appear like macrophages.

The various cell-types first mentioned may be uni- or multinucleate: usually, one or two nuclei were common among the spindle and fragment forms, whereas the larger and longer ribbon-like forms had a higher nuclear number. The nucleus was quite clear and distinct, usually containing one large nucleolus sometimes accompanied by a smaller one. Cells were seen, however, with three and four nucleoli. In some of the cultures (of upwards of 43 days in vitro) rather bizarre-shaped multinucleate cells were seen.

All forms of muscle cells may exist in a particular culture at the same time. It appears that the formation of the multinucleate ribbon-like structures is the result of growth and amitotic division without cytoplasmic cleavage. No mitotic figures could be found in any stained multinucleate cell (cf. Altschul, '46). The process by which a multinucleate ribbon, or fiber, is formed probably begins with a single fragment increasing in size, its nucleus then dividing without cytoplasmic cleavage, the whole fragment growing further to form a short ribbon-like cell, and later becoming larger and longer. These short or long ribbons can split off fragments having one or more nuclei, and these in turn can repeat the process and grow into long ribbons. These processes doubtless go on simultaneously in the culture, and anywhere along the line, any form — spindle, fragment or ribbon, meeting unfavorable conditions, may die. The average survival-time of an individual human fiber in vitro can be estimated at a month and a half.

Occasionally one could see the terminal portion of a short or long ribbon-like cell break off and form a roundish fragment. In all probability a longtudinal splitting of cells also occurs, but confirmation of this opinion requires further study. In culture no fusion of myoblasts was observed.

Isolated mononuclear cells of both large and average size could be seen in mitosis. It is not impossible that some of these could have been muscle-cells, but thus far the fate of a large mitotic cell has not been traced. For elucidating all these processes, it is hoped that growth moving-pictures can be obtained.

Striation. Longitudinal striations could be found in the ribbon-like and larger fragment forms (figs. 5, 6, 7, 9). Cross striations first became visible in the living cultures of rat muscle after an average interval of 20 days. The first striae seemed to be of a simple type: alternating light and dark bands (fig. 16). In one particular rat culture a more highly differentiated type of cross-striation, that is, having z-lines, was seen at 96 days (fig. 17). The average time for the appearance of cross-striae in human cultures was about 23 days. These seemed to have z-lines (figs. 8, 12, 13).

In both human and rat cultures, the cells may be only partially striated across their diameter, or along their length (figs. 12, 13). In some instances, in the living rat fibers, cross-striae would appear in a particular cell and later seem to disappear, but would again become visible at a subsequent observation period. In the human cultures, cross-striae were seen to disappear, but their reappearance was not noted. This was also seen in some rat cells; and in neither species was their disappearance followed by immediate disintegration of the cell.

One particular rat culture (044) was maintained continuously for more than a year and a half, and its history recorded. Observations on this culture as a whole, over a period of a year, disclosed that there were days when many good cross-striations were to be seen, alternating with other times when the striations were less clear, and with still other occasions on which they could not be found at all.

Contraction. Spontaneous contractions in tissue cultures of embryonic skeletal muscle were noted by Lewis, Friedheim, Burkhardt and Szepsenwohl. These are doubtless related to the fibrillation which characterizes denervated muscle (cf. Tower, '39). Among our adult human muscle cultures, only one instance of spontaneous contraction was observed. This was in material from a leg amputation of a 73-year-old woman. On the fifty-second day of culture, a cell devoid of cross-striae was seen to contract spontaneously, about sixteen times a minute. It continued with rests for about 20 minutes, then ceased completely.

In the adult rat, however, the spontaneous contraction of the muscle-ribbons, fragments and spindle-cells, was quite widespread. The average time for the initiation of contraction was about 15 days. These contractions exhibited great variations — all gradations from a shallow shivery kind to a deep pulsation. In many of these types of contraction the speed could vary from slow to rapid. The contraction could be steady or regular or run a pattern: a few slow deep contractions, then an increase in rate with the contractions becoming rapid and not deep, then a sudden break with a rest period, and so on, the pattern repeating.

As far as the individual fibers go, the contractions may involve only a part of the cell (one portion contracting, the other immobile) or the entire cell may be in action.

The overall contractions in a given culture varied too, at different observation periods, from many contracting fibers to none. In the case of the long-term rat culture, the fastest contraction-rate of the explant with its outgrown fibers was 220 times per minute, the slowest about 70, with the average about 150. The average rate of contraction among individual fibers of other cultures was about 138 per minute. A moving picture at low magnification was obtained of a large field of outgrowth containing fibers contracting simultaneously at different rates.

In the case of the long-term rat culture (044), observations over a period of a year have shown that it had high and low points of contractile activity, i.e., days of good contraction, or high from the standpoint of number of fibers contracting and their vigor, to low activity, or none.

Fibers with and without cross-striations were seen to contract. Thus far a constant relationship between periods of good striation and good contractility can not be established.

The culture had its best activity, or high, when it was about 5 months old, during which time there was a period of 47 days of continuous contraction. The last feeble contraction of the original explant with its budded-out fibers was observed when the culture was 1 year and 24 days old. All that remained of the original explant was a strip of fibrosed material. Nevertheless, the descendents of the isolated fibers which wandered out from the original explant are still living in this culture at 1½ years, the time of writing. Cross-striated fibers and contractions can be seen at times.

At various intervals during the life of this culture, pieces have been cut from the clot and transferred to new cover-slips. These pieces produced muscle-cells which developed, contracted and showed cross-striae. When these cultures appeared deteriorated, i.e., their cytoplasm became vacuolated, having shiny granules or blebs along the cell membrane, they would again be transferred and the pattern of the previous culture would be repeated. One such sub-plant has survived six such transfers.

Since this year-and-a-half-old culture from the region of the knee-joint is unquestionably completely denervated, yet continues to produce new muscle-buds which exhibit cross-striation and contractility, it can be said that in vitro at least, no nerve connection of any sort is essential to the multiplication of skeletal muscle fibers, or to their exercise of contractility.

It would seem that in general, washing in saline and feeding a culture stimulates it to contract. Out of 121 observations, in 83 instances there was stimulation; in 38 the culture remained unaffected.

Unsuccessful attempts have been made to induce contractions by chemical means: by doubling the amounts of calcium chloride and of potassium chloride separately in the washing saline. The potassium chloride was used only on human cultures.

It was reported by de Lustig ('42) that eserine and small concentrations of acetyl choline would activate the automatic contractility of skeletal muscle in explants from chicken embryos. This did not occur in our cultures of adult mammalian material. One to three drops of a 1/15,000 solution of eserine sulfate were added directly to rat cultures which had a previous record of contraction, but no significant increase resulted. The human cultures were unaffected in any way by the addition of the same concentration of eserine sulfate with a drop of feeding mixture. Another human culture was treated with a 1/500 dilution of eserine sulfate with no results during 15½ hours. A 1/1000 dilution of eserine sulfate together with acetyl choline chloride in the feeding mixture was tried on a human culture with no resulting contraction. After an interval of 2½ weeks the concentration was doubled. This affected the health of the fibroblasts, and 3 days later all cells including muscle were dead. Another human culture was given the 1/500 dilution of eserine sulfate together with acetyl choline chloride directly. No contraction resulted and the culture was disintegrating the next day. Rat serum was also substituted for human placental serum in feeding the human cultures for a short time (three washings) but it did not prove stimulating, or irritating.

SUMMARY

1. Adult skeletal muscle from human and rat individuals can regenerate and multiply in vitro. The multiplication is chiefly amitotic. No innervation of any sort is required for the maintenance of such a culture for as long as a year and a half.

2. Normal adult skeletal muscle-cells in both rat and human can exist in many different forms. The most common of these are spindle-shapes, round, club or rectangular fragment-types, and ribbon-like forms. Machophage-like types sometimes can be seen in the rat.

3. Both human and rat skeletal muscle-cells can contract spontaneously in vitro.

4. Cross-striations are not necessary for contraction in adult human or rat skeletal muscle and do not appear to be a stable component of muscle-cells growing in vitro.

5. Attempts to induce or increase contraction by the addition of calcium chloride or of potassium chloride to the medium, or of eserine sulfate with and without acetyl choline chloride, have failed in the concentrations used.

For most of the photographs used in this study, the authors' thanks are due to Mr. Walter I. O'Neill of the Department of Surgical Pathology.

LITERATURE CITED

Altschul, R. 1946 Nucleosis of skeletal muscle: its value as a biological test. Science, vol. 103, p. 567.

Burkhardt, L. 1936 Differenzierung von Skelettmuskulatur im Explantat. Arch. f. exper. Zellforsch., vol. 18, p. 512.

Chèvremont, M. 1940 Le muscle squelettique cultivé in vitro. Transformations d'éléments musculaires en macrophages. Arch. de biol., vol. 51, p. 313.

Favata, B. V., A. H. Dowdy, R. L. Sewell and J. G. Vincent 1944 The pathology of experimental clostridial infections in dogs. Surg., Gynec. and Obst., vol. 79, p. 660.

Forbus, W. D. 1926 Pathologic changes in voluntary muscle. Arch. Path., vol. 2, p. 318.

Freidheim, E. A. H. 1931 Morphologische und funktionelle Untersuchungen an isolierten, in vitro gesüchteten, Skelettmuskelfasern. Arch. f. exper. Zellforsch., vol. 11, p. 303, also 385.

Levi, G. 1916 Migrazione di elementi specifici differenziati in colture di miocardio e di muscoli scheletrici. Arch. per le sc. med., vol. 40, p. 1.

Lewis, M. R. 1915 Rhythmical contraction of the skeletal muscle tissue observed in tissue cultures. Am. J. Physiol., vol. 38, p. 153.

Lewis, W. H., and M. R. Lewis 1917 Behavior of cross-striated muscle in tissue cultures. Am. J. Anat., vol. 22, p. 169.

deLustig, E. Sacerdote 1942 Estudio del automatismo muscular en cultivos in vitro con eserina, acetilcolina, adrenalina y atropina. Rev. soc. argent. de Biol., vol. 18, p. 524.

Mauer, G. 1939 Die histologischen Vorgänge in verletzen Gewebe quergestreifter Musculatur und ihre Klärung mit Hilfe der Gewebekultur. Arch. f. exper. Zellforsch., vol. 23, p. 125.

Pappenheimer, A. M. 1939 The pathology of nutritional muscular dystrophy in young rats. Am. J. Path., vol. 15, p. 179.

Pogogeff, I. A., and M. R. Murray 1945 Regeneration of adult skeletal muscle in vitro. Science, vol. 101, p. 174.

De Renyi, G. S., and M. J. Hogue 1931 Studies made by means of microdissection on skeletal muscle grown in tissue culture. Arch. f. exper. Zellforsch., vol. 11, p. 304.

——— 1934 Studies on skeletal muscle grown in tissue culture. Arch. f. exper. Zellforsch., vol. 16, p. 167.

Simms, H. S., and M. Sanders 1942 The use of serum ultrafiltrate in tissue cultures for studying fat deposition and for virus-propagation. Arch. Path., vol. 33, p. 619.

Szepsenwohl, J. 1942 Las modificaciones del automatismo muscular en el desarrollo embrionario, estudiado en cultivos ''in vitro.'' Rev. soc. argent. de Biol., vol. 18, p. 71.

Tower, S. S. 1939 Reaction of muscle to denervation. Physiol. Rev., vol. 19, p. 1.

PLATE 1

EXPLANATION OF FIGURES

1 Round muscle fragment with constricted nucleus. Human, 25 days in vitro. Helly, mucicarmine. × 1120.

2 Muscle cell completing amitotic division. From same culture as figure 1.

3 Round muscle cell increasing in size. From same culture as figure 1.

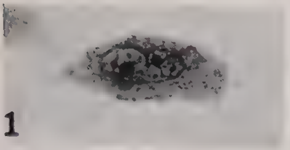
4 Elongating muscle-cell from same culture as figure 1. Longitudinal striae appearing.

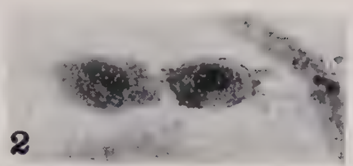
5 Club shaped muscle cell with longitudinal striations. Human, 51 days in vitro. Zenker, phosphotungstic acid haematoxylin. × 910.

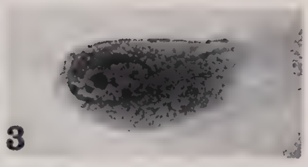
6 Binucleate fragment, with longitudinal striations. Human, 48 days in vitro. Zenker, phosphotungstic acid haematoxylin. × 560.

7 Multinucleate ribbon-forms. Human, 51 days in vitro. Zenker, phosphotungstic acid haematoxylin. × 455.

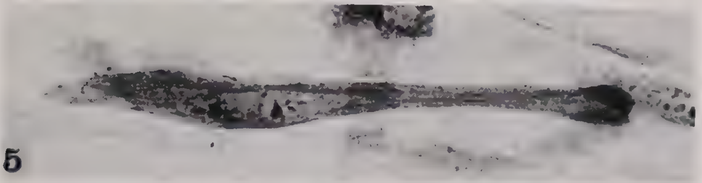
8 Cross striated ribbon. Human, 20 days in vitro. Zenker, phosphotungstic acid haematoxylin. × 980.

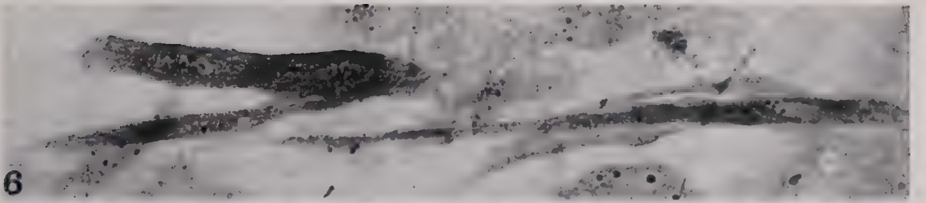

1

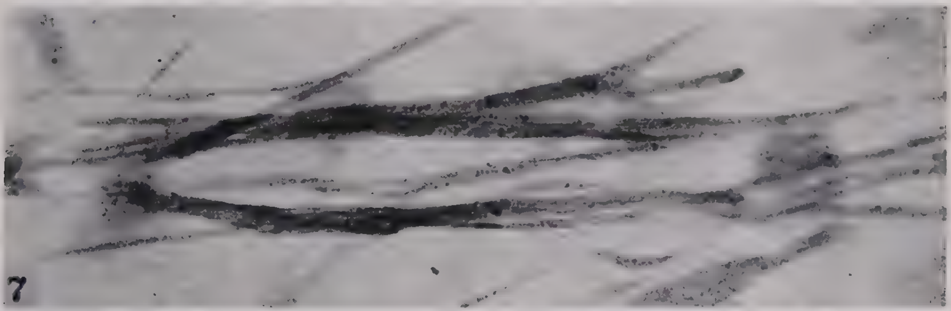

2


3

4


5


6


7

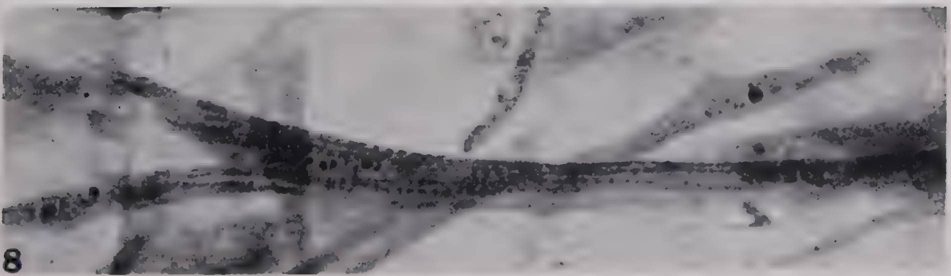

8

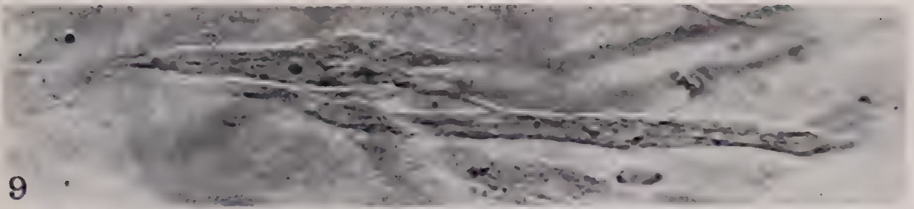
PLATE 2

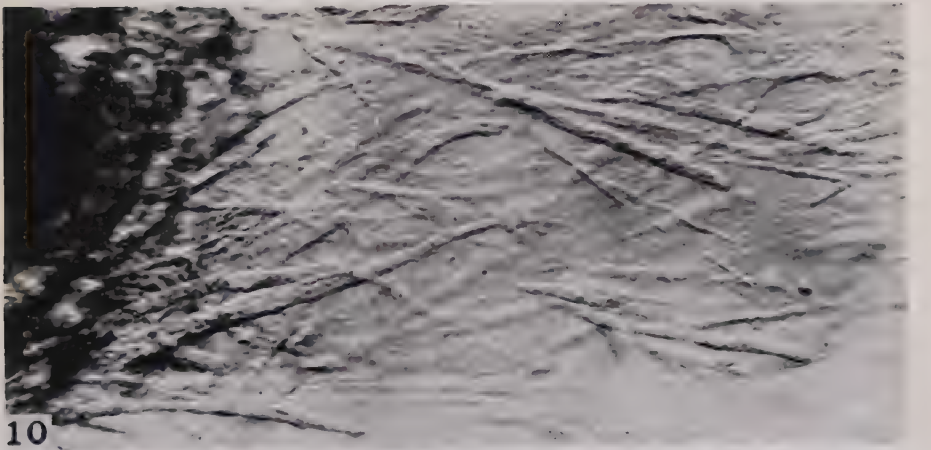
EXPLANATION OF FIGURES

9 Muscle fragments with longitudinal striae. Human, living, 27 days in vitro. × 600.

10 Muscle ribbons emerging from explant. Human, living, 27 days in vitro. × 265.

11 Ribbon forms and various fragment types. Human living, 16 days in vitro. × 265.


9


10

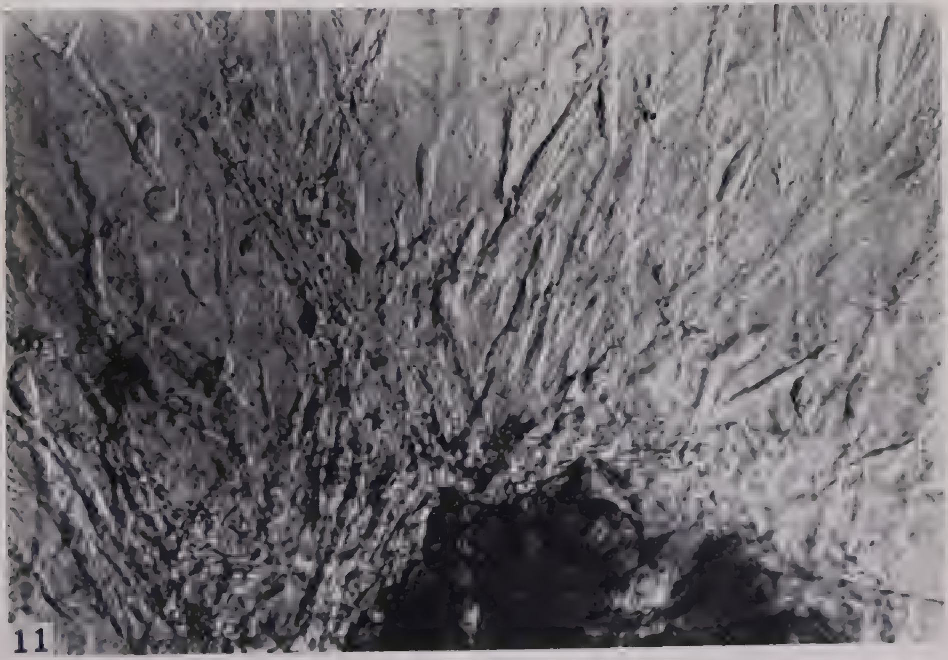

11

PLATE 3

EXPLANATION OF FIGURES

12 Cross striated muscle fragment. Human, living, 30 days in vitro. × 600.

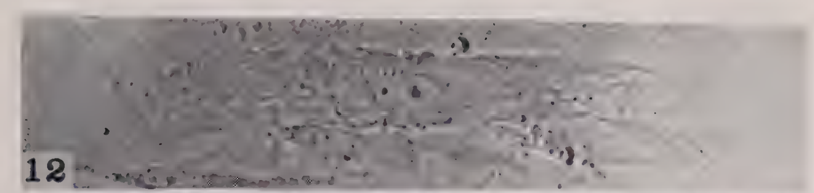
13 Same cell as in figure 12, 24 hours later. Note increase in cross-striations.

14 Trinucleate muscle-cell. Human, living, 9 days in vitro. × 265.

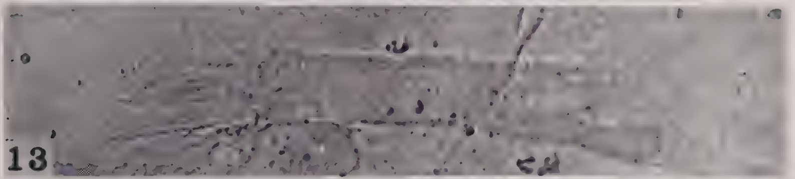
15 Long muscle ribbons. Rat, 60 days in vitro. Zenker, phosphotungstic acid haematoxylin. × 140.

16 Fiber with simple cross-striae. Rat (044) living, 67 days in vitro. × 400.

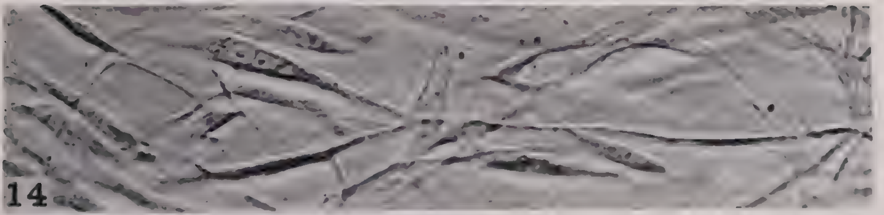
17 Fibers showing Z-lines. Rat (044) living, 96 days in vitro. × 400.



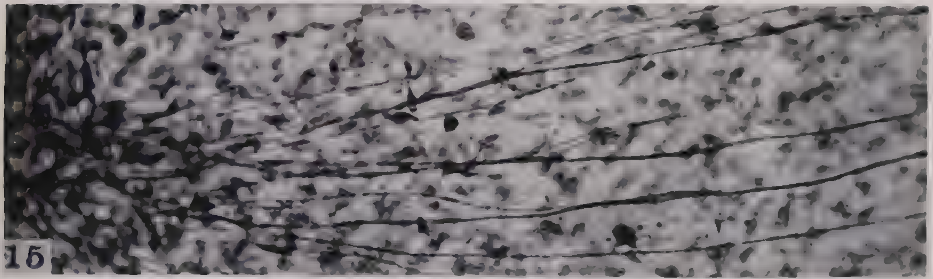
12



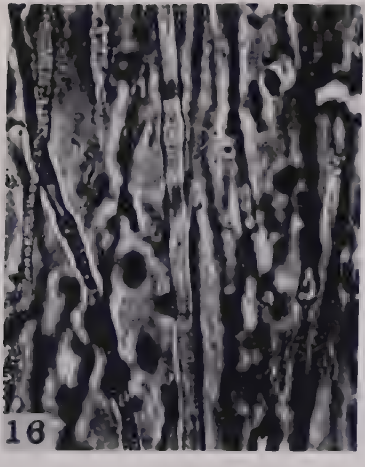
13



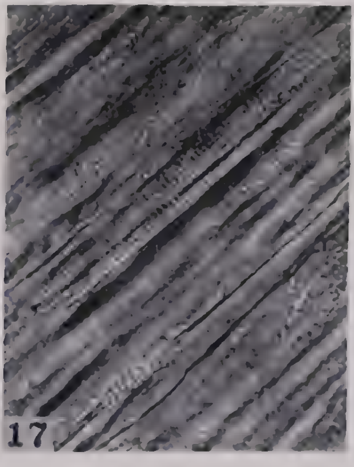
14



15



16



17

THE TIME OF OVULATION IN THE MONKEY[1]

EDMOND J. FARRIS
The Wistar Institute of Anatomy and Biology,
Philadelphia, Pennsylvania

In a recent report, a new method was described for determining the probable time of ovulation in women (Farris, '46). The test is based on the observation that the urine of a patient at about the time of ovulation will produce a hyperemia in the ovaries of an immature rat when administered to the animal subcutaneously. It is postulated that a positive reaction indicates that ovulation is taking place.

This assumption is based upon indirect evidence supported by two observations: (1) That certain relatively infertile women conceived when coitus took place during an ovulation which provided a positive urine test in rats, and (2) That no conception followed coitus in the absence of such a positive test.

In order to secure direct evidence of the occurrence of ovulation, the test was repeated on a series of monkeys whose abdomens were explored close to the time that ovulation was predicted.

MATERIALS AND METHODS

Observations were made on nine rhesus monkeys (Macaca mulatta) during one menstrual cycle in each monkey during the winter breeding season. This was made possible by cooperation of Dr. George W. Corner, Director of the Department of Embryology, Carnegie Institution of Washington, and by Mr. A. G. Rever and Mr. W. J. Cleary of the department staff. The animals were housed at the Carnegie monkey colony in Baltimore, in individual metabolism cages, equipped with containers for collecting urine. The urine of each animal was collected daily, from the fourth day of the menstrual cycle until the ovulation reaction was considered to have been completed, and was shipped daily to Philadelphia packed in dry ice.

---

[1] This investigation was aided by a grant from The Samuel S. Fels Fund.

In brief, the occurrence of ovulation was detected by the reaction of the ovary of the immature white rat to the urine of the monkey. If ovulation was not taking place, the urine had no effect upon the ovary of the rat. If it was taking place, the rat's ovaries became hyperemic.

The monkey urine passed during the night was employed. Two cm^3 were injected subcutaneously into each of two Wistar rats, between 22 and 25 days of age, and weighing between 30 and 50 gm. At the end of 2 hours the rats were killed by illuminating gas, and their abdomens were opened immediately. Each ovary was inspected, and its degree of redness was compared with the colors on the Munsell color chart (Farris, '46). When the reactions to the tests indicated that ovulation had taken place, the monkeys were explored surgically. Under nembutal anesthesia, and with full aseptic precaution, the abdomen was opened and the ovaries exposed to direct observation. Judgment as to whether ovulation had or had not occurred recently, and an estimate of the time elapsed between ovulation and exploration, was made on the basis of experience gathered by Dr. Corner in many scores of such explorations, many of which were followed by histological study of the ovaries, and by Mr. Rever during years of participation in similar studies while assistant to Dr. Carl G. Hartman (Corner, Hartman and Bartelmez, '45). The details of observations on the present cases are reported below.

RESULTS

Table 1 summarizes the observations upon eight of the monkeys. The "+" sign indicates the presence of hyperemia in the rat's ovaries, and an "0" indicates its absence. "D" signifies a doubtful hyperemia. These symbols are explained

TABLE 1

Ovulation in the monkey as confirmed by laparotomy.

MONKEY NO.	REACTIONS	DAY OF MENSTRUAL CYCLE															
		4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
708		0	+	+	+	+	0		0–L								
747		0	+	+	+	+	+	0	0–L								
723		+	D	0	0	+	+	+	+	+	0	0–L					
727	Normal	0	0	0	0	0	+	0	0	0	D	+	+	+	+	+	–L
749				0	0	+	D	0	0	0	0	+	+	+	+	+	0 –L
754		+	0	0	+	+	+	+	D	0	0–L						
720		0	0	0	+	D	+	+	D	0–L							
710	Abnormal	0	0	+	D	+	D	D	D	+	0	□-L					

□ Indicates the time interval during which ovulation occurred, as determined by inspection of the ovaries at laparotomy.

L Day of laparotomy.
+ Hyperemia.
0 No hyperemia.
D Doubtful hyperemia.

The first seven monkeys experienced normal reactions, with 4 or 5 consecutive days of hyperemia. The last one had an abnormal ovulation cycle, with interrupted days of hyperemia. It should be noted by reference to the □ that not only did ovulation in all normally reacting animals take place as indicated, but it occurred on the last day of the reaction, or within 24 hours.

in table 1 and characterize the three grades of reaction. It is believed that ovulation is completed by the end of the fifth day of color reaction. In cases with 4 days of color, however, it is probable that ovulation occurs on the day following the completion of the hyperemic reaction. These statements are based on the fact that most conceptions in women took place on the last day of the color reaction (Farris, '46).

The first seven monkeys listed in table 1 gave normal reactions; the last one had an abnormal reaction. A normal reaction is one in which a definite hyperemia appears in the rats' ovaries for 4 or 5 consecutive days. An abnormal reaction is one in which hyperemia does not appear on consecutive days.

Ovulation

Laparotomy was performed upon each of the eight animals, on the days indicated by the letter "L" in table 1. Examination of the ovaries revealed that the first seven monkeys had ovulated. The last animal, monkey 710, which gave an abnormal reaction, possessed a large unruptured follicle in its right ovary at operation. A bimanual pelvic examination of this animal the next morning indicated that ovulation had occurred.

Findings at laparotomy follow

No. 708. Exploration Feb. 15, 1945, day 11 of cycle. The right ovary contained a recently ruptured follicle, presenting a red crater which bled when wiped lightly with gauze. There was no subsurface appearance as of lutein tissue. Estimate: less than 48 hours after ovulation. The following menstrual flow began Feb. 28, 1945. (It was estimated by our method that ovulation had occurred 2 days previous to laparotomy.)

No. 747. Exploration Feb. 13, 1945, day 11 of cycle. The ovary presented a red crater which did not bleed on wiping with gauze. There was no subsurface yellow color or texture of corpus luteum tissue. Estimate: 2 to 3 days after ovulation. The next menstrual flow began Feb. 27, 1945. (It was estimated by our method that ovulation had occurred 2 days previous to laparotomy.)

No. 723. Exploration March 2, 1945, day 14 of cycle. The left ovary exhibits a crater about 1.5 mm in diameter, surrounded by a yellowish-white subsurface area 3 mm in diameter. The ovary bulges slightly but is not yet filled out as by a solid corpus luteum. Estimate: 2 to 3 days after ovulation. The next menstrual flow began March 13, 1945. (It was estimated by our method that ovulation had occurred 2 days previous to laparotomy.)

No. 727. Exploration Feb. 21, 1945, day 19 of cycle. The left ovary contained a large crater, which was bleeding spontaneously and looked very recent. There was no subsurface appearance as of lutein tissue. Estimate: 24 hours or less after ovulation. The next menstrual flow began March 2, 1945. (By our method, it was estimated that ovulation had occurred within 12 hours.)

No. 749. Exploration Feb. 19, 1945, day 19 of cycle. The right ovary presented a very fresh-looking crater, which bled lightly on wiping with gauze. There was no subsurface appearance of lutein tissue. Estimate: about 1 day after ovulation. The next menstrual flow began Feb. 28, 1945. (It was estimated by our method that ovulation had occurred within 24 hours.)

No. 754. Exploration March 3, 1945, day 13 of cycle. The right ovary bore a red stigma, about 1.5 mm in diameter. This was healed over and did not bleed on wiping with gauze. There was no subsurface color or texture as of a corpus luteum. The estimate from macroscopic examination was about 3 days after ovulation. The ovary was removed and sectioned. On microscopic examination the ruptured follicle (early corpus luteum) corresponded to the period 3–4 days after ovulation (Corner, Hartman and Bartelmez, '45). (It was estimated by our method that ovulation had occurred 2 days previous to laparotomy.)

No. 720. Exploration Feb. 17, 1945, day 12 of cycle. The left ovary presented a large red crater which did not bleed on gentle wiping with gauze. Ovary was not greatly bulged, no subsurface color or texture as or corpus luteum. Estimated: 1 to 2 days after ovulation. The next menstrual flow began March 3, 1945. (By our method, it was estimated that ovulation had occurred 1 day previous to laparotomy.)

No. 710. Exploration Feb. 15, 1945, day 13 of cycle. The right ovary contained a large unruptured follicle, or nearly mature size (estimated 5–6 mm diameter). On the following day ovulation was diagnosed by digital bimanual palpation of the ovary. The following menstrual flow began March 5, 1945. (It was estimated by our method that ovulation had occurred 1 day previous to laparotomy.)

Post-ovulatory interval

The lengths of the intervals between ovulation and the start of the next menses of six animals, as checked by both the ovulation test and laparotomy, are shown in table 2. These intervals varied from 9 to 17 days. Ovulation occurred from cycle days 8 to 19, in cycles ranging from 23 to 29 days in length.

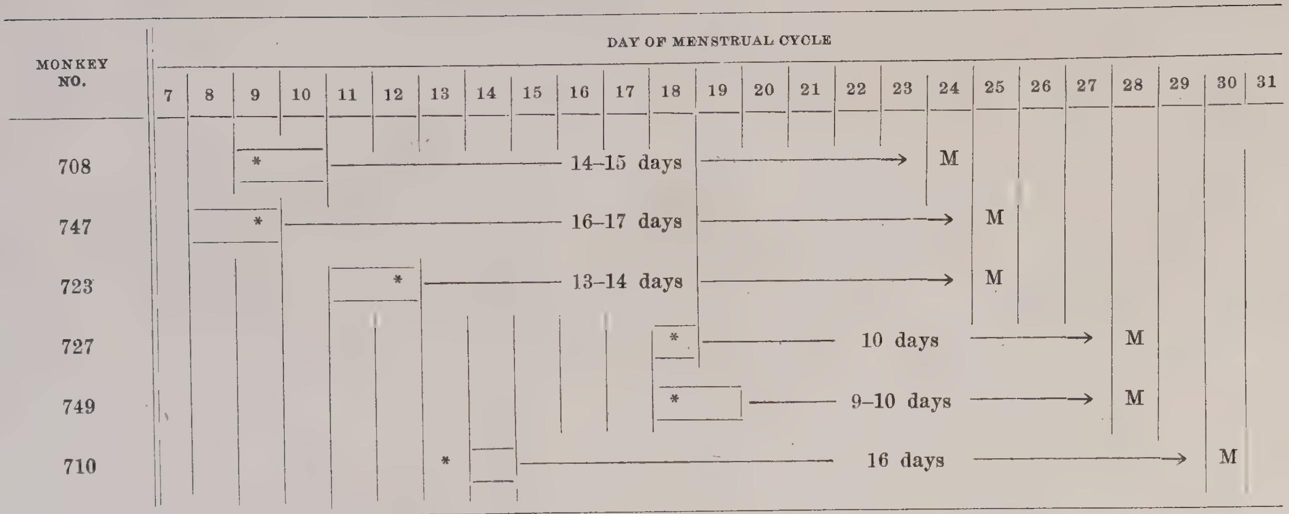
Anovulatory cycles

Some monkeys that were tested gave no reaction. An example of such an experience occurred in the case of monkey 228. Samples of the urine of this animal were tested on days believed to be 4 through 18. The reaction was reported as positive only on day 4. In view of the fact that no test was positive during the remaining 13 days, a tentative diagnosis of an anovulatory cycle was made, and an exploratory laparotomy was performed. This monkey proved to be pregnant. Further checking revealed that the usual vaginal bleeding (''placental sign'') associated with pregnancy was thought, by the attendant, to be a normal menstrual flow. It was determined that the 1 day of positive color appeared between the twenty-second and twenty-seventh day of the cycle.

In addition, three other monkeys, the urine of which failed to produce hyperemia in preliminary tests, were considered

TABLE 2

The post-ovulatory interval.



* Ovulation predicted by the rat test.

▭ Ovulation confirmed by laparatomy.

M Menstruation day 1.

Showing the post-ovulatory interval in six monkeys. Each animal was tested for ovulation time and subjected to laparotomy at the time of ovulation. The interval varied from 9 to 17 days.

anovulatory, the diagnosis being based upon bimanual rectal pelvic examination.

DISCUSSION

The present observations indicate that the urine of the monkey at the time of ovulation produces hyperemia of the rat ovary. Such a reaction to the urine of normal women lasts only 3 or 4 days, whereas in the case of the monkey it continues for 4 or 5 days. It has been assumed, in the case of women, that ovulation occurs on the final day of the color reaction. This hypothesis is based on the observation that conceptions have occurred when intercourse took place on this day.

On the basis of the condition of the corpus luteum of the monkey at exploratory laparotomy, and of the occurrence of conception in those women who planned coitus in relation to the time of normal ovulation reactions, it is concluded that the test described is diagnostic of the time of ovulation in both women and monkeys.

If hyperemia fails to appear in the rat's ovaries after injection of the monkey's urine at the time that ovulation should occur, it is postulated that the cycle is anovulatory. This statement is based upon the observation on monkey 228, as well as those on the three other monkeys which were considered anovulatory.

In the case of women (Farris, '46) whose urine gave no reaction, conception failed to occur. In the case of monkeys, both laparotomy and bimanual examination of the ovaries proved that ovulation failed to take place.

A word of caution for those who attempt to repeat the work. Rectal palpation of the monkey usually induces a positive reaction at any time of the cycle. The collected urine should be kept cold, to avoid a toxic response in the rats after injection.

SUMMARY AND CONCLUSIONS

1. A test which appears to indicate the time of ovulation was made upon a series of rhesus monkeys (Macaca mulatta).

2. In all of the seven animals in which the test was positive for 5 successive days, ovulation was shown by laparotomy to have occurred at the end of this period.

3. In all of the four animals in which the reactions were absent, laparotomy in one case and bimanual examination in three others failed to show ovulation.

4. In one monkey in which the sequence of the test reactions was abnormal, laparotomy showed a large mature follicle in one of the ovaries and bimanual examination revealed that ovulation had occurred by the next morning.

Appreciation is expressed to Drs. George W. Corner, Clarence J. Gamble and Douglas P. Murphy, for suggestions incorporated in this paper.

LITERATURE CITED

FARRIS, E. J. 1946 A test for determining the time of ovulation and conception in women. Am. J. Obstet. and Gynec., (in press).

CORNER, G. W., C. G. HARTMAN AND S. W. BARTELMEZ 1945 Development, organization and breakdown of the corpus luteum in the rhesus monkey. Carnegie Inst. Wash. Publ. 557, Contrib. to Embryol., vol. 31, pp. 117–146.

AN ANOMALOUS MIDDLE MENINGEAL ARTERY

FRANK N. LOW
Department of Anatomy, School of Medicine, West Virginia University,
Morgantown, West Virginia

ONE FIGURE

The surgical importance of the middle meningeal artery has led to a number of studies of the details of its course. Chandler and Derezinski ('35) have reported its variations within the middle cranial fossa in about 1200 cases, and quote data concerning its origin and distribution in 450 other cases previously reported by Kanavel and Davis, Bartlett, and Yatsuta. Toida ('34) has reported similar data from 122 adult Chinese skulls. The practical interest in the middle meningeal artery centers around its ramifications in the middle cranial fossa, particularly on the inner surface of the squamous portion of the temporal bone where its branches obstruct the point of entry in operations on the semilunar ganglion. The case herein reported is believed to be of interest because the abnormal origin and course of the artery differs so greatly from the normal that such an anomaly, surgically encountered, would be very difficult to interpret unless such a condition were previously known to exist.

The middle meningeal artery normally arises from the first or mandibular portion of the internal maxillary artery between the lateral (external) pterygoid muscle and the sphenomandibular ligament. It ascends to the base of the skull, passing between the two roots of the auriculotemporal nerve. It enters the middle cranial fossa through the foramen spinosum in the sphenoid bone and bifurcates into anterior and posterior branches within the fossa. The ramifications here have been reported in detail by Chandler and Derezinski ('35). A feature of its ramification of particular interest to this case is the occasional twig from the anterior branch which enters the orbit through the superior orbital fissure or through a small foramen in the greater wing of the sphenoid bone and anastomoses with the lacrimal branch of the ophthalmic artery. The foramen through which this twig passes is usually unnamed but is designated "foramen meningoorbitale" by Rauber-Kopsch ('14).

DESCRIPTION

The middle meningeal artery herein described was encountered in a dried adult skull in which the sutures and dentition indicated early middle age. Aside from the anomalous artery the skull possessed no unusual features except for unusually large foramina throughout. The anomaly was bilateral, the artery entering the middle cranial fossa from the orbit, through the foramen meningoorbitale which was located on both sides about 2 mm lateral to the apex of the superior orbital fissure. The foramen spinosum was absent. Close examination of the foramen ovale revealed no irregularity in its outline or any indication of arterial branches radiating therefrom. From its point of entry the artery turned laterad and bifurcated within 5 cm on both sides into upper and lower branches, apparently homologous respectively with the anterior and posterior branches of normal arteries. The upper, or anterior branch entered a bony canal (fig. 1, A) present bilaterally in 41% of cases according to Chandler and Derezinski ('35), and then pursued a normal course. The lower, posterior branch bifurcated on the right side after passing from the greater wing of the sphenoid bone to the squamous portion of the temporal bone, both branches running horizontally dorsad almost parallel to each other along the uppermost portion of the temporal bone. On the left side the posterior branch remained unbranched and ran dorsad about 1 cm from the upper edge of the squamous portion of the temporal bone. The anterior branch bifurcated just before entering its bony canal and sent a branch dorsad which ran above the posterior branch and supplied the area covered by the upper ramus of the posterior branch on the right side. The terminal distribution of the artery on both sides was normal except for an unusual branch of the anterior division on the right side in the anterior cranial fossa (fig. 1, A).

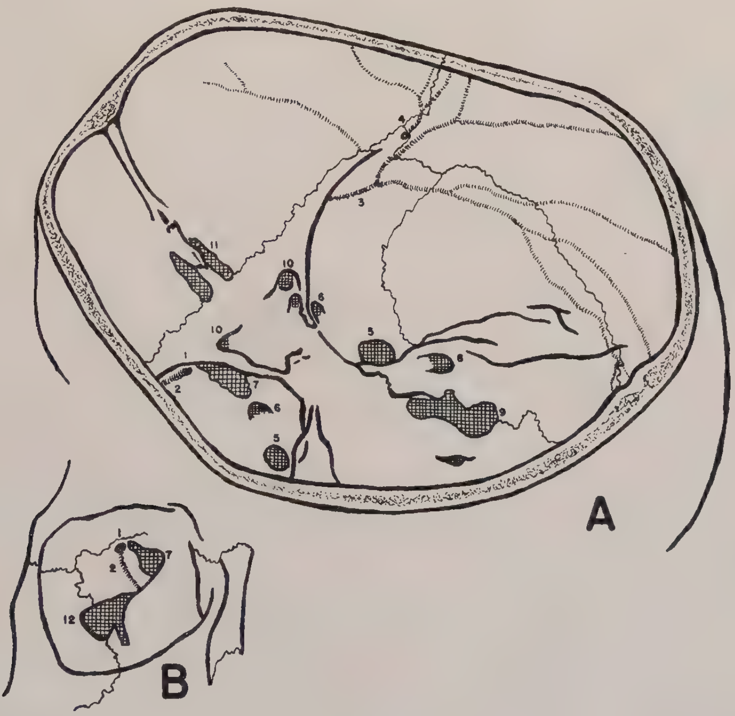


Fig. 1 (A) Postero-lateral aspect of skull with calvarium removed, from above. Some sutures, particularly those of the temporal bone, have been indicated. The grooves caused by the middle meningeal artery and its branches are represented. (B) Anterior aspect of the right orbital cavity of same skull. The groove caused by the passage of the middle meningeal artery across the great wing of the sphenoid bone is indicated. Legend: 1, foramen meningoorbitale; 2, main trunk of middle meningeal artery; 3, posterior branch of middle meningeal artery; 4, anterior branch of middle meningeal artery emerging from bony canal; 5, foramen ovale; 6, foramen rotundum; 7, superior orbital fissure; 8, internal auditory meatus; 9, foramen jugulare; 10, optic foramen; 11, cribriform plate; 12, inferior orbital fissure.

When traced backwards into the orbit the middle meningeal artery on both sides was observed to have grooved the anterior surfaces of the great wings of the sphenoid bones (fig. 1, B) lateral to the superior orbital fissure from the foramen meningoorbitale to the sphenomaxillary fissure. It could not be traced backward beyond its entrance into the orbit.

DISCUSSION

The probable origin of this anomalous middle meningeal artery is from the third or sphenomaxillary portion of the internal maxillary artery since its entrance into the sphenomaxillary fissure is distant from the normal point of origin. If this anomalous artery arose in a normal position it would have had to run parallel to the internal maxillary artery for about 3 cm to reach the sphenomaxillary fissure, an unlikely circumstance. If the origin were from the third portion of the internal maxillary artery then an attempt at extracranial ligation of the middle meningeal artery at the point of normal origin would probably result in failure to discover the artery. Its course across the great wing of the sphenoid bone in the orbit would, in this case, present an easier approach.

Numerous variations in the origin of the middle meningeal artery have been reported. The most commonly observed variation is its entrance through the foramen ovale. According to Chandler and Derezinski ('35) this occurs in nearly 1% of cases while presence of both foramina with interconnection is somewhat more frequent. The origin of the artery from the complex of vessels in the middle ear, including the stapedial, has been reported to have occurred bilaterally by Altmann ('32). Of greater interest here is the fact that in one of the specimens of Chandler and Derezinski ('35) the anterior branch of the middle meningeal artery was observed to arise bilaterally from the orbit. Toida ('34) reported this to have occurred in five instances in 122 skulls. This condition probably represents doubling of the middle meningeal artery, the posterior branch arising from the usual place in the internal maxillary artery and entering through the foramen spinosum, the anterior branch arising from the third portion of the internal maxillary artery and entering through the orbit. In one of Toida's cases ('34) both anterior and posterior branches came from the orbit. This is the only similar case encountered in nearly 1900 cases reviewed. The association of this anomalous artery with the foramen meningoorbitale is probably only incidental since its course through the spheno-maxillary fissure rules out the possibility of origin from the lacrimal branch of the ophthalmic.

The intracranial course of the artery herein described is of interest because of the abnormally high ramification of the posterior branch along the inner surface of the squamous portion of the temporal bone. In no place is the lower part of the inner surface of the squamous portion of the temporal bone traversed by the artery. The surgical approach to the semilunar ganglion would likely not encounter any of its branches. On the other hand, it would be difficult to ligate its main intracranial trunk since this is located under the margin of the lesser wing of the sphenoid bone where it forms the upper anterior margin of the middle cranial fossa instead of on the floor of the middle cranial fossa within easy access.

LITERATURE CITED

ALTMANN, F. 1932 Über zwei seltene Anomalien der Arteria meningea media. Ztschr. f. d. ges. Anat. (Abt. 1), vol. 97, pp. 509–514.

CHANDLER, S. B., AND C. F. DEREZINSKI 1935 The variations of the middle meningeal artery within the middle cranial fossa. Anat. Rec., vol. 62, pp. 309–319.

RAUBER-KOPSCH 1914 Lehrbuch der Anatomia des Menschen. Abt. 3, Leipzig.

TOIDA, N. 1934 Ueber die Endocranischen Furchen der Arteria Meningea Media bei den Chinesen. J. Orient. Med., vol. 21, pp. 13–14.

THE SEQUENCE OF EPIPHYSIAL UNION IN THE OPOSSUM

S. L. WASHBURN
*Department of Anatomy, College of Physicians and Surgeons,
Columbia University, New York*

The present study was undertaken with the hope that the understanding of epiphysial union in a generalized marsupial might elucidate the origin of the pattern of mammalian growth. The opossum is particularly suitable as a subject for such a study because of its primitive nature. McCrady ('38), after reviewing the various theories concerning the evolutionary position of the opossum, concludes that "one point on which almost all the evidence is in agreement is that the Didelphyidae are the most primitive marsupials alive today." Simpson ('44) describes the morphological differences between modern and some Cretaceous opossums as slight, although 60,000,000 years of evolution occurred between them.

The primitive position of the opossum among the marsupials seems clearly established. However, there is less agreement concerning the relations of the marsupials to the placental mammals. The most recent opinion (Simpson, '45) is that both groups are descended from the Triassic pantotheres. Whether the opossums are viewed as directly ancestral or in a collateral position, their primitive nature gives insight into the early phases of mammalian evolution which is obtainable in no other way.

The possession of epiphyses is a mammalian character (Romer, '33), and the sequence of union of the various epiphyses has been claimed to be a basic mammalian character (Stevenson, '24). Now that many more data are available, it is clear that there is more variation in the age-order of union than was at first supposed. Differences in epiphysial sequence among eight primates are illustrated in Schultz's ('44) monograph on the gibbon.

The sequences of epiphysial union of the placental mammals fall into two major divisions; (1) Those in which union is delayed into extreme old age, and (2) those in which union takes place in the first half of the animal's life. The first group is composed of rat (Dawson, '25, '27) and guinea pig (Zuck, '38) and, probably, many other rodents. The second group contains the remainder of the placental mammals. The importance of the first group is far greater than the number of included animals would suggest because it contains the common laboratory animals used in the majority of experimental investigations of growth. An appreciation of the peculiarities of growth in rats, mice, and guinea pigs is of fundamental importance to those who are using these animals in studies on growth, nutrition, or aging. Additional contrast between the two fundamental types is provided by the fact that the regional groupings of epiphyses, which are so characteristic of most mammals, are not so well defined in these rodents, particularly in the guinea pig.

It has been shown (Washburn, '43) that the epiphyses of one region (elbow, wrist, knee, hip, etc.) unite to their diaphyses at much more nearly the same time than epiphyses in general. If changes in the sequence of union occur, usually whole regions are accelerated or decelerated, rather than individual epiphyses. Grouping of the epiphyses greatly reduces the number of variables which must be considered, so that generic or familial differences and evolutionary trends may be easily appreciated.

For example, in the orang-utan union in the hand (epiphyses of the phalanges and metacarpals) and wrist (distal radius and ulna) is delayed compared to man (Schultz, '41). What appears to be a number of differences when sequences of ungrouped epiphyses are compared proves to be delayed union in hand and wrist, probably associated with late growth in these regions in the orang-utan.

The primary objectives of this study of the opossum are: (1) The determination of the time — relations of epiphysial union, and (2) regional analysis of the sequence of epiphysial union. In the opossum, is union delayed into extreme old age? Are regional groupings of the epiphyses discernible?

MATERIAL AND METHODS

The thirty opossums used in this study were all supplied by The Wistar Institute of Anatomy and Biology. The writer is greatly indebted to the Institute and to its Director, Dr. E. J. Farris, for the loan of these valuable specimens. Four skeletons had already been prepared for study by Dr. J. A. McClain of Villanova College, to whom the author wishes to express his thanks. The skeletons were prepared by maceration as the presence of any ligaments or cartilage makes the accurate assessment of the degree of epiphysial union difficult. Three genera of opossums are represented in the series: Didelphys, the large Virginia opossum, and two small, tropical opossums from Panama, Metachirus and Philander.[1]

The ages of the opossums were supplied by The Wistar Institute and are based on the length of time which each animal was in captivity plus an estimate of its age at the time it was received. In the case of the Virginia opossums the estimates are undoubtedly extremely accurate. There may be a somewhat wider margin of error in the case of the tropical opossums, eleven of whom were adult when received at the Institute. The ages in months are given in table 1. The skeletons were arranged in order of ascending maturity, and the order of epiphysial union recorded, as given in table 1. The order is based on completed union, for the reasons given in an earlier publication (Washburn, '43). When the skeletons are compared in detail, it is possible to make much finer judgments than can be recorded in a small table. In places the order in table 1 is based on partial union and small differences observable on the skeletons. The preference given coracoid over acetabulum, or fibula distal over tibia distal, depends on

---

[1] The nomenclature is that suggested by Goldman ('20).

TABLE 1

The sequence of epiphysial union in three genera of opossums.

GENUS AND AGE IN MONTHS				GIRDLE		ELBOW				FEET			S.	WRIST		HIP			ANKLE		KNEE		
Didelphys	Philander	Metachirus	TOTAL NUMBER	Coracoid	Acetabulum	Ulna proximal	Humerus distal	Medial epicondyle	Radius proximal	Calcaneus	Phalanges	Metapodials	Humerus proximal	Radius distal	Ulna distal	Lesser trochanter	Greater trochanter	Femur head	Fibula distal	Tibia distal	Fibula proximal	Tibia proximal	Femur distal
12, 16 28	11	13	7	U	U	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o
11	14, 22		5	U	U	U	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o
13, 20 20, 21			4	U	U	U	U	U	o	o	o	o	o	o	o	o	o	o	o	o	o	o	o
24			1	U	U	U	U	U	o	U	U	o	o	o	o	o	o	o	o	o	o	o	o
	16	22	2	U	U	U	U	U	o	U	U	U	o	o	o	o	o	o	o	o	o	o	o
26			2	U	U	U	U	U	U	U	U	U	o	o	o	D	o	o	o	o	o	o	o
27			1	U	U	U	U	U	U	U	U	U	U	o	o	D	D	D	o	o	o	o	o
	27	29	2	U	U	U	U	U	U	U	U	U	U	U	o	o	o	o	o	o	o	o	o
	41	29	2	U	U	U	U	U	U	U	U	U	U	U	U	U	U	o	o	o	o	o	o
	18	32	2	U	U	U	U	U	U	U	U	U	U	U	U	U	U	o	U	U	o	o	o
	29	19	2	U	U	U	U	U	U	U	U	U	U	U	U	U	U	o	U	U	U	U	U

o = Open; U = Completed Union; D = Completed Union in Didelphys only.
S = Shoulder Region.

such small differences. The order in such cases means very little, and it is more fruitful to consider the epiphyses which are bracketed in table 1 as uniting at approximately the same time (subject to qualifications to be considered later).

DISCUSSION

The relationship of epiphysial union to age will be considered first, subsequently the regional analysis of the sequence given in table 1, and lastly the evolutionary significance will be treated.

In no animal in this series was the process of epiphysial union complete. The youngest animals were adult with completed dentition, and the series includes the oldest opossums ever kept at The Wistar Institute. Yet even the oldest animals are not osteologically mature. In the opossum the process of epiphysial union does not even commence until an age at which it would be completed in the cat (Shapiro, '30).

The maturation of the mammalian skeleton follows a plan which may be outlined as follows: first, primary parts of the vertebrae (body and arch) unite; second, the primary parts of the girdles (coracoid, and acetabulum); third, the epiphyses in the extremities join the diaphyses; fourth, the epiphyses of the girdles and axial skeleton fuse (iliac crest, ischial tuberosity, vertebral border of the scapula, vertebral epiphyses). Even in the two most mature opossums of this series the head of the femur has not united to the neck, and in no case are the epiphyses of the girdles and vertebrae united.

The opossum, then, clearly is similar to the rat (Dawson, '25, '27) and guinea pig (Zuck, '38) in which epiphysial union, even in the extremities, is delayed into extreme old age and may never be completed. This appears to be a truly primitive condition, reminiscent of the reptiles in which growth may continue into old age. It is unfortunate that the animals most commonly used in the study of growth and aging possess the primitive characters of delayed union and long-continued growth, which contrast so markedly with the sharply defined period of growth characteristic of the vast majority of placental mammals.

Although there is a correlation between skeletal maturity and chronological age, there are marked exceptions. Considering only the Virginia opossums, the oldest animal is among the least mature osteologically. The teeth of this specimen are worn, and it would be judged to be one of the oldest animals on the basis of cranial characters. There are other irregularities of a similar nature, and, although the series is small, it seems safe to conclude that chronological age bears less relation to epiphysial union in the opossum than in any mammal so far reported. If man varied as much as the opossum, normal adults of over 30 years of age might have a skeletal maturity appropriate to 14 or 15 years.

Not considering the three most exceptional cases, it is possible to give an approximate statement of the time of epiphysial union. The primary parts of the girdles (coracoid, acetabulum) must unite near the end of the first year,[2] during the second year the epiphyses of the elbow region and feet join, and, in the third year the remainder of the epiphyses of the extremities fuse.

Turning from chronological considerations to the regional analysis of the sequence of epiphysial union, the group of epiphyses which constitute the regions are bracketed in table 1. The regional order is: elbow, feet, shoulder, wrist, hip, ankle, knee. Maturation in the hip region is much later in the tropical opossums than in Didelphys. It is important to note that unions of the lesser trochanter, greater trochanter, and femur head are all earlier in the Virginia opossums. The entire hip region has accelerated. It is precisely because groups of epiphyses shift in this manner that a regional concept is so useful in comparing the sequences of epiphysial union of various mammals.

In some regions the epiphyses fuse at practically the same time. In these opossums the epiphyses of the ankle and knee regions are either all open or all united. In the wrist, radius distal only slightly precedes ulna distal. However, radius proximal unites considerably later than the other epiphyses of the elbow region, and placing it with the others might be considered arbitrary if the opossum was the only animal considered. However, in monkeys (Washburn, '43), the proximal epiphyses of the ulna and radius fuse at nearly the same time, and in man (Stewart, '34) the medial epicondyle is the last epiphysis of the elbow region to unite. Comparison of many sequences shows that the epiphyses of each region unite at much more nearly the same time than epiphyses in general and that maturity tends to be accelerated or decelerated in a whole region, rather than in a bone or an epiphysis.

---

[2] Through the work of Hartman ('28), McCrady ('38), and Moore and Bodian ('40) a great deal is known about the development of the opossum during the first 100 days. More data are needed concerning growth during the latter part of the first year to fill the gap between these studies and the period at which epiphysial union commences.

The hip region illustrates the same situation. The lesser trochanter, greater trochanter, and femur head unite at nearly the same time in Didelphys, but femur head is delayed and less closely associated with the trochanters in the tropical opossums. In monkeys the order is lesser trochanter, femur head, greater trochanter. The order of fusion within the region changes, but the same cluster of epiphyses is easily recognizable, and its position relative to other similar clusters may be traced throughout the mammals.

Certain epiphyses of the opossum differ morphologically from those of other mammals. The ossification center of the medial epicondyle of the humerus joins with the epiphysis formed by the fusion of trochlea and capitellum. Even the small center of the lateral epicondyle is included. The single, composite epiphysis unites to the diaphysis. In placental mammals the medial epicondyle remains separate and fuses to the diaphysis without first joining the composite epiphysis of the articular region.

A similar, but less pronounced, difference is seen in the hip region. The epiphyses of the head of the femur, lesser and greater trochanters are more closely associated than in placental mammals.

Table 2 gives regional sequences of a variety of mammals for comparison with that of the opossum. Data were lacking concerning the time of union of the epiphyses of the hands and feet in some cases. Therefore, the comparison is limited to the order of six regions. The most important point to note is that the same regions, the same clusters of epiphyses, are easily recognized in a great variety of mammals. In fact one of the most fundamental and all pervasive features of mammalian growth, found from opossum to man,[3] is that the factors determining growth exercise a regional control. This is essentially a "field concept," having much in common with Butler's theory of dental evolution (discussed by Dahlberg, '45). The conception that growth is controlled by regions, rather than bone by bone or epiphysis by epiphysis, is of evolutionary importance. If changes in proportions can only be achieved by a large number of mutations affecting individual bones, the chances that so many beneficial changes will become the possession of any one population are fantastically small. However, if the same result can be attained by a single change in one region, the possibility of the rapid acquisition of favorable mutations is increased. For example, a single change in the growth rate of the knee region will produce the same effect as correlated changes in the femur, tibia, and fibula.

The difficulties inherent in viewing adaptive changes as being due to a large number of independent mutations have long been recognized (Hooton, '31). A regional or field concept of growth-controlling factors gives a theoretical framework which is compatible both with genetics and comparative anatomy. Closely related mammals often differ in the proportions of their limb bones. Here is a theory which will account for such differences with a minimum amount of genetic change.

Although the possession of the same regional groupings of epiphyses is a basic mammalian character, the sequence of union is not. Comparison of the sequences in table 2, shows that the elbow region is the only one which is always in the same position. Inclusion of a greater variety of animals and of the epiphyses of the feet, would increase the variation. For example, according to Todd and Todd ('38) union occurs last in the hip region in elephants. Also, according to the same authors, the epiphyses of the phalanges unite extremely early in ungulates (sheep) and late in the sirenians.

---

[3] The guinea pig (Zuck, '38) appears to be the only mammal whose sequence of epiphysial union does not lend itself to regional analysis.

There appears to be no general evolutionary trend in the changes of position of the various regions. In so far as the six regions shown in table 2 are concerned, lemur and man are identical and even the bison is more like man than is the gorilla.

TABLE 2

Sequences of epiphysial union of a variety of mammals analyzed by regions.

OPOSSUM	HEDGEHOG [1]	LEMUR (Todd, '30)	MONKEY (Washburn, '43)
Elbow	Elbow	Elbow	Elbow
Shoulder	Hip	Hip	Hip
Wrist	Ankle	Ankle	Ankle
Hip	Wrist	Knee	Wrist
Ankle	Knee	Wrist	Knee
Knee	Shoulder	Shoulder	Shoulder

RAT (Dawson, '25)	GORILLA (Randall, '44)	BISON (Koch, '35)	MAN (Stewart, '34)
Elbow	Elbow	Elbow	Elbow
Ankle	Hip	Ankle	Hip
Hip	Shoulder	Hip	Ankle
Knee	Ankle	Knee	Knee
Wrist	Knee	Wrist	Wrist
Shoulder	Wrist	Shoulder	Shoulder

---

[1] Based only on seven specimens of the hedgehog, Erinaceus, at the Museum of Comparative Zoology of Harvard University. Fortunately, the ages are such that a tentative order can be constructed from these few specimens.

SUMMARY AND CONCLUSIONS

1. The sequence of epiphysial union in three genera of opossums is presented (table 1).
2. Epiphysial union commences after the dentition is complete near the end of the first year. The process continues throughout the second and third years and is not completed, even in the oldest opossums.
3. The regional pattern of epiphysial union, characteristic of the majority of mammals, is present in the opossum.

4. The evolutionary significance of the pattern is discussed.
5. Epiphysial union takes place first in the elbow region in all mammals. Otherwise there are many differences in the sequence of union, and no general evolutionary trends are discernible.

LITERATURE CITED

DAHLBERG, A. A. 1945 The changing dentition of man. Jour. A.D.A., vol. 32, pp. 676–690.

DAWSON, A. B. 1925 The age order of epiphyseal union in the long bones of the albino rat. Anat. Rec., vol. 31, pp. 1–17.

——— 1927 Further studies on the epiphyses of the albino rat skeleton, with special reference to the vertebral column, ribs, sternum, and girdles. Anat. Rec., vol. 34, pp. 351–363.

GOLDMAN, E. A. 1920 Mammals of Panama. Smithsonian Misc. Collections, vol. 69, no. 5, pp. 1–309.

HARTMAN, C. G. 1928 The breeding season of the opossum (Didelphis virginiana) and the rate of intrauterine and postnatal development. J. Morphol., vol. 46, pp. 143–215.

HOOTON, E. A. 1931 Up from the Ape. Macmillan Company, New York.

KOCH, W. 1935 The age order of the epiphyseal union in the skeleton of the European bison (Bos bonasus L.). Anat. Rec., vol. 61, pp. 371–376.

MCCRADY, E. JR. 1938 The embryology of the opossum. Am. Anat. Memoirs, no. 16, pp. 1–226.

MOORE, C. R., AND D. BODIAN 1940 Opossum pouch young as experimental material. Anat Rec., vol. 76, pp. 319–327.

RANDALL, F. E. 1944 The skeletal and dental development and variability of the gorilla. Human Biology, vol. 16, pp. 23–76.

ROMER, A. S. 1933 Vertebrate Paleontology. Univ. of Chicago Press, Chicago.

SCHULTZ, A. H. 1941 Growth and development of the orang-utan. Contrib. Embryol., no. 182, Carnegie Inst. Wash., vol. 29, pp. 57–100.

——— 1944 Age changes and variability in gibbons. Am. J. Phys. Anthrop., n.s., vol. 2, pp. 1–129.

SHAPIRO. H. H. 1930 Growth and time correlations between ossification centers in the long bones and calcification centers in the mandibular dentition of the cat. Internat. J. Orthodontia, vol. 16, pp. 690–702.

SIMPSON, G. G. 1944 Tempo and Mode in Evolution. Columbia Univ. Press, New York.

——— 1945 The principles of classification and a classification of mammals. Bull. Am. Mus. Nat. Hist., vol. 85, pp. 1–350.

STEVENSON, P. H. 1924 Age order of epiphyseal union in man. Am. J. Phys. Anthrop., vol. 7, pp. 53–93.

STEWART, T. D. 1934 Sequence of epiphyseal union, third molar eruption, and suture closure in Eskimos and American Indians. Am. J. Phys. Anthrop., vol. 19, pp. 433–452.

TODD, T. W. 1930 Comparative Youth. The physical aspect. Child Development, vol. 1, pp. 79–89.

TODD, T. W., AND A. W. TODD 1938 The epiphyseal union pattern of the ungulates with a note on Sirenia. Am. J. Anat., vol. 63, pp. 1–36.

WASHBURN, S. L. 1943 The sequence of epiphysial union in Old World monkeys. Am. J. Anat., vol. 72, pp. 339–360.

ZUCK, T. T. 1938 The age order of epiphyseal union in the guinea pig. Anat. Rec., vol. 70, pp. 389–399.

APPROVED MOTION PICTURE FILMS

The following films have been approved by one or more members of the Review Committees of the American Association of Anatomists or the American Society of Zoologists, and The Wistar Institute:

SUBJECT AND TITLE	LENGTH IN REELS (1–16 MM. REEL = 400 FT. RUNNING TIME ABOUT 15 MIN.)	PRODUCER	CONDITIONS FOR SECURING FILMS	DISTRIBUTOR
ANATOMY				
Congenital anomalies (monochrome, silent) (kodachrome, silent)	1	J. Sarnoff	Sale: Monochrome, $25.00 Kodachrome, $75.00	Sarnoff Motion Picture Film Library 1406 Albemarle Road Brooklyn, N. Y.
* The management of cicatricial strictures of the esophagus. Treatment of cancer of the esophagus. Bronchoscopy with insufflation of sulfanilamide in chronic bronchiectasis (kodachrome, silent)	1	E. H. Ingersoll	Rental, $8.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* Surgical anatomy (kodachrome, silent)	4	C. J. Baumgartner	Sale Kodachrome	Billy Burke Productions 7416 Beverly Blvd. Hollywood, Calif.
BLOOD CELLS				
* 10. Normal and abnormal white blood cells in tissue cultures (monochrome, silent)	1	W. H. Lewis M. R. Lewis A. R. Rich M. M. Wintrobe	Rental, $4.00 per day Sale, $25.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
CANCER CELLS				
* 6. Dividing cancer cells in vitro (monochrome, silent)	160 ft.	W. H. Lewis M. R. Lewis	Rental, $2.00 per day Sale, $10.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* 3. Tumor cells and macrophages in tissue cultures. Rat sarcomas and carcinomas (monochrome, silent)	360 ft.	W. H. Lewis	Rental, $3.50 per day Sale, $20.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
CIRCULATION				
* The control of small blood vessels (monochrome, silent)	1	G. P. Fulton B. R. Lutz	Rental or sale	Dr. G. P. Fulton College of Liberal Arts Boston University Boston, Mass.
* Effect of various drugs on contractions of mesenteric lymphatics in the rat (monochrome, silent)	300 ft.	R. L. Webb	Rental, $3.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.

SUBJECT AND TITLE	LENGTH IN REELS (1–16 MM. REEL = 400 FT. RUNNING TIME ABOUT 15 MIN.)	PRODUCER	CONDITIONS FOR SECURING FILMS	DISTRIBUTOR
Circulation (Cont.)				
* The heart and circulation (monochrome, silent)	1	A. J. Carlson	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
Mesenteric lymphatics, their conduct and the behavior of their valves in the living rat (monochrome, silent)	1	R. L. Webb	Rental, $4.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* Micromanipulative studies of blood capillaries (monochrome, silent)	1	B. W. Zweifach	Rental, $4.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* 7. Growth of capillaries and endothelium from the sub-cutaneous tissue of 7-day chick embryos in 2-day cultures in Locke-bouillon-dextrose medium (monochrome, silent)	140 ft.	W. H. Lewis	Rental, $2.00 per day Sale, $9.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
CYTOLOGY				
* 8. Pinocytosis. Drinking by cells (monochrome, silent)	264 ft.	W. H. Lewis	Rental, $3.00 per day Sale, $16.50	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* 9. Dividing normal adult rat fibroblasts in vitro (monochrome, silent)	160 ft.	W. H. Lewis	Rental, $2.00 per day Sale, $10.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
DIGESTION				
* Digestion of foods	1	A. J. Carlson H. G. Swann	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
* Alimentary tract	1	A. J. Carlson H. G. Swann	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
* Our teeth	1	John R. McCrory Studios	Rental Sale	Knowledge Builders 625 Madison Ave. New York, N. Y.
EMBRYOLOGY				
* Activity of heart muscle, from rat embryos, grown in tissue culture (monochrome, silent)	1	C. M. Goss	Rental, $4.00 Sale, $25.00	Dr. C. M. Goss University of Alabama University, Ala.
Development of a salamander (monochrome, silent)	1	L. S. Stone and T. C. Kramer	Rental Sale	Films, Inc. 330 W. 42nd St. New York, N. Y.
* 1. The early development of the rabbit egg in vitro (monochrome, silent)	1	W. H. Lewis and P. W. Gregory	Rental, $4.00 per day Sale, $25.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.

SUBJECT AND TITLE	LENGTH IN REELS (1–16 MM. REEL = 400 FT. RUNNING TIME ABOUT 15 MIN.)	PRODUCER	CONDITIONS FOR SECURING FILMS	DISTRIBUTOR
Embryology (Cont.)				
* 4. The early development of the rabbit egg (short film — selected scenes from the long film) (monochrome, silent)	80 ft.	W. H. Lewis and P. W. Gregory	Rental, $1.50 per day Sale, $5.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* 5. Early divisions, 2–8 cells, of living monkey egg in vitro (monochrome, silent)	80 ft.	W. H. Lewis and C. G. Hartman	Rental, $1.50 per day Sale, $5.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
Forming an egg (kodachrome, sound)	1	D. C. Warren and H. M. Scott	Rental, $3.00 per day	Poultry Department Kansas State College Manhattan, Kans.
* Where chick life begins (kodachrome, silent)	1	A. L. Romanoff	Rental Sale	Ralston Purina Co. St. Louis, Mo.
* 11. Development of Zebra fish egg (monochrome silent)	1	W. H. Lewis and E. C. Roosen-Runge	Rental, $4.00 per day Sale, $25.00	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* Development of the opossum (kodachrome silent)	150 ft.	E. J. Farris	Rental, $3.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
ENDOCRINOLOGY				
* Endocrine glands	1	A. J. Carlson H. G. Swann	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
EXCRETION				
* Work of the kidneys	1	A. J. Carlson H. G. Swann F. J. Mullin	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
GENERAL				
Practical preventive orthodontics (kodachrome silent)	2	M. J. Futterman	Rental	Dr. M. J. Futterman 1749 Grand Concourse Bronx, N. Y.
* High speed motion pictures taken with stroboscopic light (monochrome, silent)	1	H E. Edgerton K. J. Germeshausen H. E. Grier	Rental free Sale, $25.00	Prof. Charles E. Locke Massachusetts Institute of Technology Cambridge, Mass.
* Seeing the unseen (monochrome, silent)	1	H. E. Edgerton	Rental free Sale, $25.00	Prof. Charles E. Locke Massachusetts Institute of Technology Cambridge, Mass.
* Tropical opossums (kodachrome, silent)	1	E J. Farris	Rental, $8.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* Unseen worlds (monochrome, sound)	1	RCA Victor Camden, N. J.	Transportation charges only	Wm. J. Ganz Co. 19 E. 47 St. New York, N. Y.

SUBJECT AND TITLE	LENGTH IN REELS (1–16 MM. REEL = 400 FT. RUNNING TIME ABOUT 15 MIN.)	PRODUCER	CONDITIONS FOR SECURING FILMS	DISTRIBUTOR
General (Cont.)				
* The eyes of science (monochrome, silent)	3	Bausch & Lomb Optical Co.	Rental free	Bausch & Lomb Optical Co. Rochester, N. Y.
* Heredity	1	H. Strandskov	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
NERVOUS SYSTEM				
* Effect of electrical stimulation of the right ansiform lobe of the cerebellum of the cat (Felis domestica) (monochrome, silent)	100 ft.	S. L. Clark	Rental	Dr. Sam L. Clark Department of Anatomy Vanderbilt University Nashville, Tenn.
* The nervous system (monochrome, silent)	14	I. V. Pavlov and others	Rental or sale	Brandon Films, Inc. 1600 Broadway New York, N. Y.
* How the eye functions	1	John R. McCrory Studios	Rental Sale	Knowledge Builders 625 Madison Ave. New York, N. Y.
* How the ear functions	1	John R. McCrory Studios	Rental Sale	Knowledge Builders 625 Madison Ave. New York, N. Y.
REPRODUCTION				
* The biology of reproduction in rats (kodachrome, silent)	1	E. J. Farris	Rental, $8.00 per day	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
* Maternal behavior in the rat (monochrome, silent)	350 ft.	F. A. Beach	Rental, $8.00 per day Sale, $30.00	American Museum of Natural History New York, N. Y.
Mating behavior in the leopard frog, Rana pipiens (monochrome, silent)	250 ft.	F. A. Beach	Rental, $8.00 per day Sale, $20.00	American Museum of Natural History New York, N. Y.
Ovulation in the frog, Rana pipiens (monochrome, silent)	512 ft.	R. Rugh	Rental, $5.00 per day Sale, $25.00	Dr. Roberts Rugh N. Y. University Washington Square College New York, N. Y.
* Proestrous and oestrous behavior in the guinea pig (monochrome, silent)	150 ft.	W. C. Young E. W. Dempsey R. Hertz and H L. Myers	Rental, $1.50 per day Sale	The Wistar Institute 36th St. and Woodland Ave. Philadelphia, Pa.
A rapid pregnancy test (kodachrome, silent)	250 ft.	U. J. Salmon and others	Rental free	Dr. U. J. Salmon 875 Fifth Avenue New York, N. Y.
* Studies in human fertility	1 (2000 ft.)	Audio Productions, Inc.	Rental free	Ortho Products, Inc. Linden, N. J.
* Reproduction among mammals	1	Melvin Brodshaug Frederick T. Howard Herluf H. Strandskov	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.
RESPIRATION				
Mechanisms of breathing (monochrome, sound)	1	V. Johnson	Sale	Encyclopaedia Britannica Films, Inc. 20 N. Wacker Drive Chicago 6, Ill.

---

* Copy deposited in the Film Depository of The Wistar Institute, available to all scientists for use at The Wistar Institute.

THE ACTION OF THIOUREA ON THE INTRACELLULAR COLLOID OF THE THYROID GLAND

RAFAEL GRASSO[1]

Institute of General Anatomy and Embryology, Faculty of Medicine of Buenos Aires, Argentina and Laboratory of Histology of the Faculty of Medicine of Montevideo, Uruguay

THREE PLATES (TWELVE FIGURES)

Since the work of Kennedy ('42), Richter and Clisby ('42), Mackenzie and Mackenzie ('43), Astwood and col. ('43) it has become well known that thiourea and related compounds produce a great hypertrophy of the thyroid gland, together with an inhibition of its function. The histological changes found, consisted of a progressive increase in height of the thyroid cells, congestion, decrease of the staining properties, reabsorption of the follicular colloid, and cellular hyperplasia with formation of foldings and papillae of the epithelium. In spite of these signs of hyperactivity these drugs produce a real diminution of the thyroid function.

In spite of the great progress in the study of the action of these drugs on the thyroid from the point of view of experimental physiology, biochemistry and therapeutics, the study of the cytological changes has been neglected. Thomas ('44) made a cytological study of the secretion antecedent of the gland with the method of Bensley ('16) and found an increase of the basal colloid which he interprets as being due to the paralysis of the basal secretion, and which may be explained by the action of the drug on the permeability of the cellular membrane.

---

[1] Fellow from Uruguay supported by the Argentine National Commission of Culture.

THE ANATOMICAL RECORD, VOL. 95, NO. 4
AUGUST, 1946

With the help of the freezing-drying method (Gersh, '32), De Robertis ('41–'42) was able to demonstrate the presence of the intracellular colloid and to study the cytological processes of secretion and reabsorption of the follicular colloid. Furthermore De Robertis and Del Conte ('44) by counting the number of colloid droplets in the cells, developed a highly sensitive method of assaying, cytologically, the degree of activity of the gland in relation to the thyreotropic factor of the pituitary gland.

In this work we have applied the above mentioned method in order to study the changes of the intracellular colloid and to determine them quantitatively.

METHODS

A total of twenty-eight white rats from 60 to 95 gm in weight were used. A group of four rats were sacrificed 1 hour after the intraperitoneal injection of 10 mg of thiourea. In other groups the drug was mixed with the drinking water with the exception of three rats which received thiouracil by mouth for 162 days.

The animals were killed with a blow on the head and the thyroids extracted rapidly, immersed in liquid air and afterwards dehydrated in high vacuum at − 30°C. Proteins of the gland were denatured in 100% alcohol for 48 hours and the thyroids embedded in nitrocellulose. The sections were stained as recommended by De Robertis ('41). The amount of intracellular colloid was calculated by the determination of the "cytological coefficient" (De Robertis and Del Conte, '44). The technique consists in counting the number of colloid droplets in ten or more activated follicles, and this amount is related to their diameters in microns. The quotient multiplied by 100 expresses the quantity of colloid in a follicle of 100 microns of diameter (Cyt. coef. $= \frac{\text{No. of colloid droplets} \times 100}{\text{Medium follicular diameter}}$).

RESULTS

1. Intracellular colloid of the thyroid gland 1 hour after injection of thiourea. In five rats killed 1 hour after the injection of 10 mg of thiourea, no histological and cytological changes could be observed. As in the normal gland (figs. 1 and 6) the follicles situated in the central part of the gland were more active than the peripheral, with cubic or cylindrical cells which seldom have few droplets in the apical poles. Droplets of basal colloid (Bensley, '16) are very rarely found; they are large, pale and granular due to the formation of ice crystals by freezing. As in the normal, the follicular colloid is abundant, the material homogeneous and darkly stained. The cytological coefficient is also unchanged, the averages being between 11.91 and 13.04 for the controls.

2. Intracellular colloid of the thyroid gland after 7, 13 and 14 days of treatment with thiourea. As seen in figures 2 and 7, changes of the thyroid gland are clearly observed after a week of administration of 0.5% thiourea in the drinking water. The intracellular colloid increases greatly so that the coefficient reaches 28.57. This coefficient rises with the time and with the doses of the drug, reaching a maximum at 13 days with 1% of thiourea and showing a value of 92.05 (figs. 3 and 8).

The size of the thyroid follicles is diminished in the first week because of the partial reabsorption of the colloid but it becomes much greater than in the normal after some time (fig. 8).

After a week of thiourea treatment, the thyroid cells almost double their height and after 13 days reach a maximum of three to four times the size of the normal (fig. 8). At this time the colloid of the central follicles is mostly reabsorbed (fig. 3) and the lumen appears as clefts filled with a pale colloid, frequently with ice-crystals; while the follicles of the periphery have a more abundant and deeper stained colloid. In some cases the reabsorption of the colloid originates pseudofollicular diverticula which frequently have a deeper stained colloid and lower epithelium.

Relatively frequently, follicles with macrophages in their lumina are found (fig. 11). These macrophages are round or elongated and have the cytoplasm loaded with colloid droplets.

These cells which very seldom appear in normal glands probably intervene in the reabsorption of the colloid.

At first (1 week) the intracellular colloid droplets are darkly stained but in time (13–14 days) they become paler and frequently show a pale central region surrounded by a deeper stained outer limit.

The basal colloid increases with the treatment and also reaches a maximum at 13 days. At this time almost every follicle shows two or three big droplets of pale colloid in the basal pole (fig. 12).

3. *Intracellular colloid of the thyroid gland after 18 days of thiourea.* In a group of five rats treated with 1% thiourea in the drinking water the cytological coefficient was 82.89, so that it diminished in comparison to the rats treated in the same way for 13 days.

The follicles are completely irregular in shape with foldings and papillae of the epithelium invading the lumen. This is reduced to very narrow clefts because of the almost complete reabsorption of the colloid (figs. 4 and 9). At this time also the more peripheral follicles have reabsorbed the colloid completely and the section of the gland shows a more homogeneous aspect. The height of the cells is very much increased and it corresponds to three to four times the height of normal cells. The congestion of the gland reaches its maximum at this time (fig. 4).

4. *Intracellular colloid of the thyroid gland after 162 days of treatment with thiouracil.* Three rats were treated "per os" with 0.02 gm of thiouracil daily during 75 days and afterwards with 0.04 gm of thiouracil during 77 days. Although this material cannot be strictly compared with the other, it is very interesting in that in spite of the high dose of thiouracil given over a very long period, the number of the colloid droplets was much lower (cytological coefficient 25.46) and the histological aspect of the gland looked more like the normal (figs. 5 and 10). At this time the congestion has almost disappeared and, in addition to the follicles with little colloid, many have abundant and well stained colloid (fig. 10). The

cells, in spite of being higher than the normal (fig. 6), are much lower than at 13 or 18 days of treatment. The connective tissue which did not show changes in the other groups appears in these rats slightly increased.

DISCUSSION

The work of Kennedy ('42), Mackenzie and Mackenzie ('43), Astwood and col. ('43), etc., clearly demonstrates that thioureas produce a hypertrophy of the thyroid gland with inhibition of the secretion of thyroid hormone. Several authors have shown that the inhibitory effect of these drugs is produced immediately after their administration. Keston and col. ('44) found that thiourea acting "in vitro" suppresses the formation of diiodotirosine and thyroxine by the thyroid tissue; De Robertis and Moura Gonçalvez ('45) found a decrease of the oxidoreduction potential immediately after the injection of thiourea or when this drug acts "in vitro"; Chagas, De Robertis and Coucero ('45) found that the penetration of radioactive iodine is almost suppressed with the injection of thiourea, and Dempsey ('44) and De Robertis and Grasso ('45) showed that thioureas have an inhibitory effect on the peroxidase activity of the thyroid gland both "in vitro" and "in vivo."

These previous results could lead to the supposition that thioureas could have an immediate effect on the cytological coefficient of the thyroid (an expression which relates the number of colloid droplets to the diameter of the follicles). As shown by De Robertis and Del Conte ('44) this is strongly and rapidly affected by other factors like thyreotropic hormone and is the most sensitive test of the activity of the gland. However, the experiments described here and in another paper (Grasso, '45) show that this coefficient is not affected 1 hour after the injection of thiourea.

Still more interesting seems the fact that when the action of this drug persists during days and weeks the coefficient, instead of decreasing, shows a progressive increase, reaching a maximum of 92.05 at 13 days. As this change of the cytological coefficient is normally an expression of the hyperactivity of the gland, even more sensitive than the reabsorption of the colloid or the height of the thyroid cells, etc. it seems rather paradoxical that simultaneously there is a real inhibition of the function of the gland. This could be explained partially by the interrelationship between the thyroid and the pituitary gland. It is generally admitted that the primary inhibition of the thyroid gland, which produces a drop in the output of the hormone, stimulates the secretion of more thyreotropic factor from the pituitary gland, thus producing the secondary hypertrophy of the thyroid. This increase of the thyreotropic factor, generally admitted, in spite of the fact that it could not be demonstrated (Gordon and col., '45), has been recently found by us (Grasso and De Robertis, '45), using the cytological method developed by De Robertis and Del Conte ('44) for the assay of the circulating thyreotropic hormone of the pituitary gland.

Our results are against the hypothesis proposed by Thomas ('44), namely that thiourea paralyses the basal secretion by changing the permeability of the cellular membrane. This drug, instead of inhibiting the secretion and reabsorption of the colloid, stimulates both processes, as shown by the increase in the intracellular colloid. The increase in the basal colloid can be interpreted as a sign of a more intense reabsorption and secretion towards the base.

If, according to these results, the thyroid cells are really producing more secretion, we have to admit, in order to explain the inhibitory effect of thiourea on thyroid function, that secretion is deficient. In other words it is lacking or very poor in thyroxine.

In fact, recent studies with the radioactive iodine and on the peroxidase activity of the gland, lead to the conclusion that thioureas act by inhibiting the process of iodinization, thus preventing the formation of diiodotirosine and thyroxine. This effect could be due to the inhibition of the peroxidase system found in the thyroid cells and, which is probably connected with the liberation of iodine from iodides (Dempsey, '44; De Robertis and Grasso, '45).

The synthesis of the protein substrate on which thyroxine is fixed is probably due to the action of proteolytic enzymes (Salter and Lerman, '36). This substrate without thyroxine, probably represents the intracellular colloid found in rats treated with thiourea. In this connection it is interesting that thiourea does not inhibit "in vitro" the proteolytic activity of the thyroid gland, while it is strongly inhibted by iodine (De Robertis and Nowinski, '45). This fact may explain why thiourea does not diminish the cytological coefficient, and also does not interfere with the increase in intracellular colloid, which is induced by the thyreotropic hormone that is secreted in excess by the pituitary gland. The fact that thiourea does not interfere with the action of thyreotropic hormone on the intracellular colloid of the thyroid (Grasso, '45) also supports this interpretation.

Summarizing, we may say that thiourea acts on the thyroid by inhibiting the process of iodinization, but it does not affect the synthesis of the protein group which is biologically inactive. The lack of thyroxine stimulates the output of thyreotropic hormone from the pituitary gland, thus inducing all the biological and cytological changes found in the thyroid. Under this action the gland is really secreting in excess but the product of secretion has less hormonal activity.

SUMMARY

The intracellular colloid of the thyroid gland of white rats was studied with the freezing-drying method, both in normal cases and in animals treated with thiourea. The injection of this drug does not change, after 1 hour, either the histological structure, or the amount of intracellular colloid measured by the cytological coefficient (De Robertis and Del Conte, '44). Thiourea supplied with the drinking water, produces with time, an increase of the intracellular colloid. The cytological coefficient (number of colloid droplets per follicle of 100 microns of diameter) increases from 13.04 in the normal, to 92.05

after 13 days of 1% thiourea in the water. In longer periods the coefficient diminishes (82.89 after 18 days). In three rats treated during 5 months with thiouracil the cytological coefficient was only 26.24. The basal colloid increases together with the coefficient, reaching a maximum at 13 days.

Other histological changes like congestion, reabsorption of the colloid, height of the epithelium, and hyperplasia are also described. It is interesting that after 5 months of treatment the gland reaches a structure more similar to the normal.

The mechanism of action of thiourea on the thyroid gland is discussed. The conclusion is reached that the cytological changes are due to the thyreotropic hormone secreted in excess by the pituitary gland. Under this action thyroid cells are really more active, as shown by the cytological coefficient, but it is supposed that the product secreted has a very low hormonal activity due to the lack of thyroxine.

ACKNOWLEDGMENT

I wish to express my sincere gratitude to Dr. E. De Robertis under whose guidance and advice this work was carried out.

LITERATURE CITED

Astwood, E. B., J. Sullivan, A. Bissell and R. Tyslowitz 1943 Action of certain sulfonamides and of thiourea upon the function of the thyroid gland of the rat. Endocrinology, vol. 32, p. 210.

Astwood, E. B., and A. Bissell 1944 Effect of thiouracil on the iodine content of the thyroid gland. Endocrinology, vol. 34, p. 282.

Bensley, R. R. 1916 The normal mode of secretion in the thyroid gland. Am. J. Anat., vol. 19, p. 37.

Chagas, C., E. De Robertis and A. Coucero 1945 Penetration of radioactive iodine in the thyroid gland colloid. Tex. Rep. Biol. Med., vol. 3, p. 170.

Dempsey, E. W. 1944 Fluorescent and histochemical reaction in the rat thyroid gland at different states of physiological activity. Endocrinology, vol. 34, p. 27.

De Robertis, E. 1941 The intracellular colloid of the normal and activated thyroid of the rat studied by the freezing-drying method. Am. J. Anat., vol. 68, p. 317.

——— 1942 Intracellular colloid in the initial stages of thyroid activation. Anat. Rec., vol. 84, p. 125.

De Robertis, E., and E. Del Conte 1944 Método citológico para determinación de la hormona tireotropa de la hipófisis. Rev. Soc. Arg. Biol., vol. 20, p. 88.

De Robertis, E., and R. Grasso Peroxidase activity of the thyroid gland under normal and experimental conditions. Endocrinology (in press).

De Robertis, E., and J. Moura Gonçalvez 1945 The oxidation-reduction potential of the thyroid follicle in normal and experimental conditions. Endocrinology, vol. 36, p. 245.

De Robertis, E., and W. W. Nowinski 1945 La actividad proteolítica de la glándula tiroides normal y patológica. Rev. Soc. Arg. Biol., vol. 21, p. 121.

———— The mechanism of the therapeutical action of iodine on the thyroid gland. (In press.)

Gersh, I. 1932 The Altmann technique for fixation by drying while freezing. Anat. Rec., vol. 53, p. 309.

Gordon, A. S., E. D. Goldsmith and H. A. Charipper 1945 Thyreotropic hormone content of the blood sera and pituitary glands of thiourea, -sulfadiazine-treated and thyroidectomized rats. Endocrinology, vol. 36, p. 53.

Grasso, R. Estudios sobre la hormona tireotropa con el método citológico. I. Acción de la hormona tireotropa y de la tiourea sobre el coeficiente citológico. (En prensa.)

Grasso, R., and E. De Robertis Estudios sobre la hormona tireotropa con el método citológica. II. La hormona tireotropa circulante en ratas tratadas con tiourea. (En prensa.)

Kennedy, T. H. 1942 Thioureas and goitrogenic. Nature, vol. 150, p. 233.

Keston, A. S., E. D. Goldsmith, A. B. Gordon and H. A. Charipper 1944 Effect of thiourea upon metabolism of iodine by rat thyroid. J. Biol. Chem., vol. 152, p. 241.

Mackenzie, C. J., and J. B. Mackenzie 1943 Effect of sulfonamides and thioureas on the thyroid gland and basal metabolism. Endocrinology, vol. 32, p. 185.

Richter, C. P., and K. H. Clisby 1942 Toxic effects of the bitter-tasting phenylthiocarbamide. Arch. Path., vol. 33, p. 46.

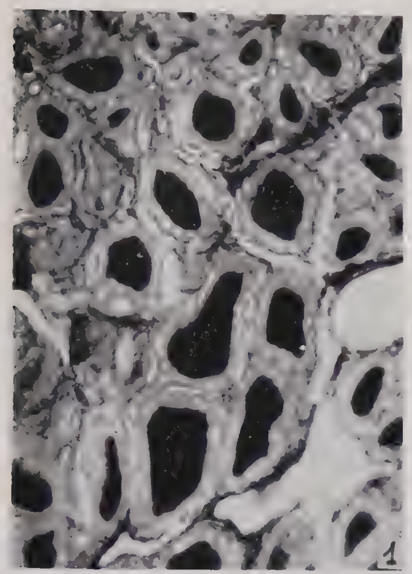
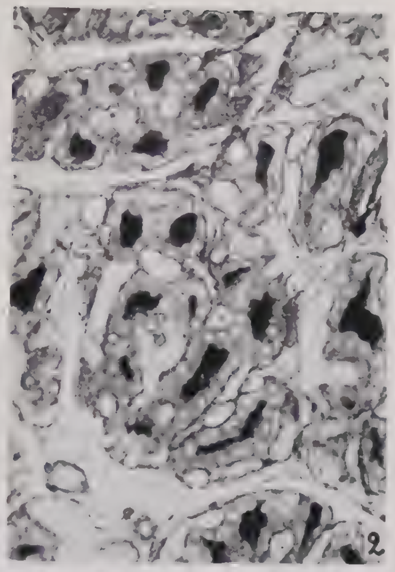
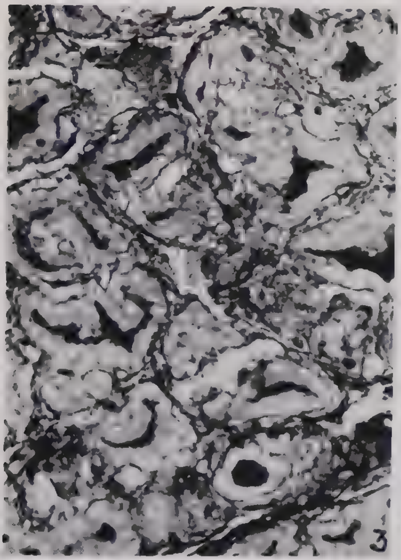
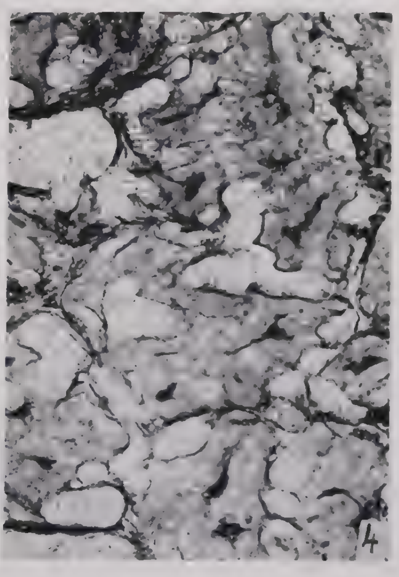
Salter, W. T., and J. Lerman 1936 The genesis of thyroid protein: clinical assays of artificial thyroid protein in human mixedema. Endocrinology, vol. 26, p. 300.

Thomas, C. L. 1944 A cytological study of the secretion antecedents of rat thyroids activated by thiourea. Anat. Rec., vol. 89, p. 461.

PLATES

EXPLANATION OF PLATES

Figures 1–12 are microphotographs of thyroid glands fixed with the freezing-drying method and stained with anilin blue-orange G. Figures 1–5 obj. 8 mm, oc. Homal II, camm. length 65 cm. Figures 6–12: 90 × obj. 6 × oc., 50 cm camm.-length. Pancromatic plate, red filter.

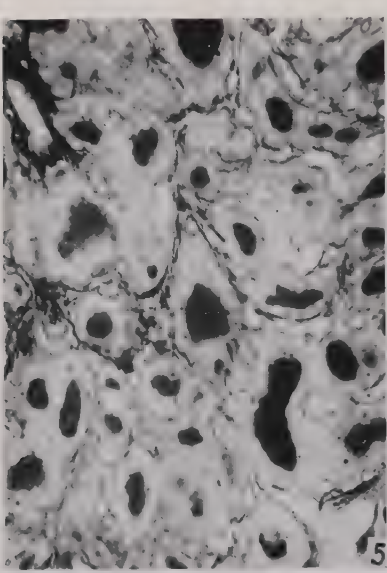





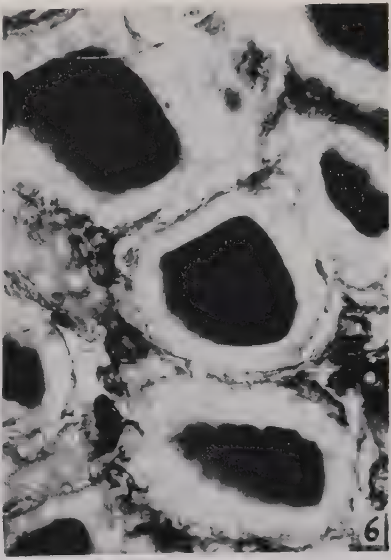
1 Thyroid gland of the normal white rat.

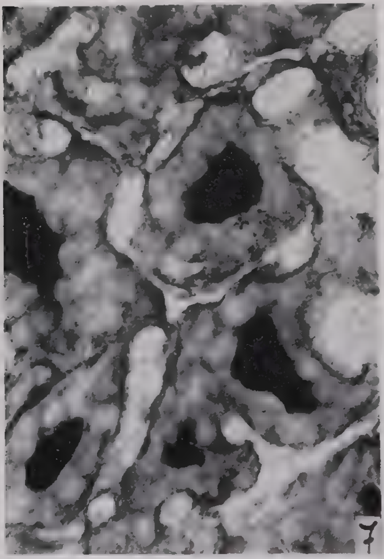
2 Thyroid gland of a rat treated with 0.25% thiourea in the drinking water during 7 days. Colloid is in reabsorption, thyroid cells are higher and have more colloid droplets.

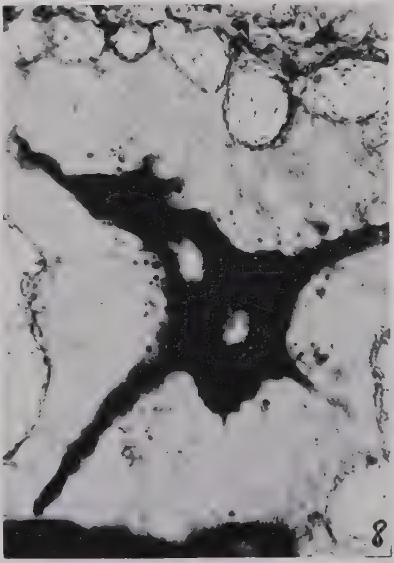
3 Thyroid gland of a rat treated with 1% thiourea during 13 days. Follicles are very hyperplastic with little colloid and very high epithelium.

4 Thyroid gland of a rat treated with 1% thiourea during 18 days. Colloid almost completely reabsorbed, enormous congestion.







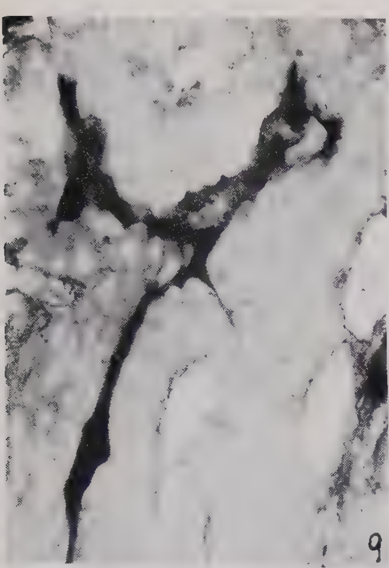


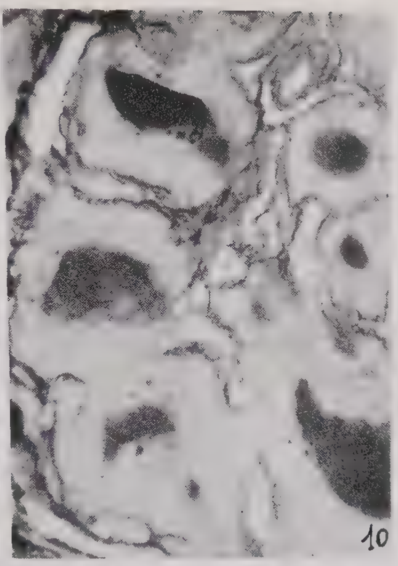
5 Thyroid gland of a rat treated with thiouracil for 162 days. The structure of the gland has returned to a more normal aspect. Some follicles are filled with colloid, no congestion.

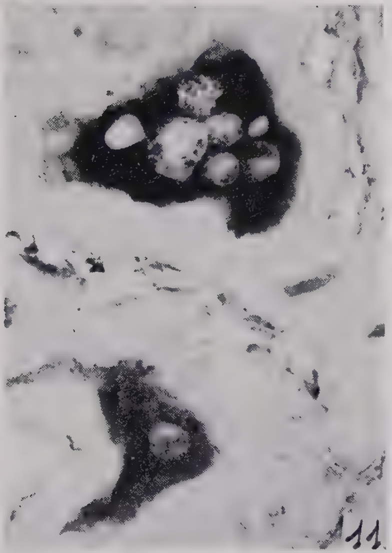
6 Thyroid gland of a normal rat. Only very few colloid droplets are seen in the cells.

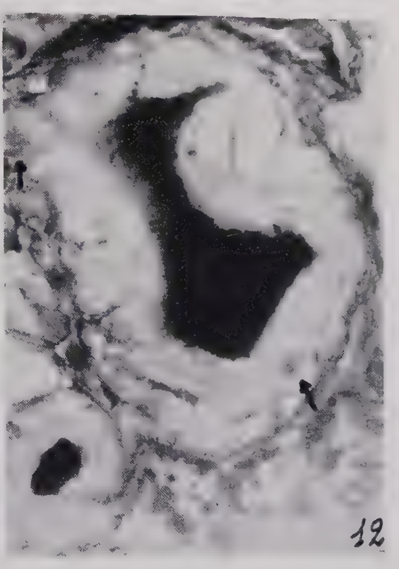
7 Thyroid gland of a rat treated with 0.25% of thiourea during 7 days. Less follicular colloid, higher epithelium with more colloid droplets, mild congestion.

8 Thyroid gland of a rat treated with 1% of thiourea during 13 days. Follicles are so greatly enlarged that only a part of one is taken in the microphotograph. Many colloid droplets are seen in the apical zone of the cells.


9


10


11


12

9 Thyroid gland of a rat treated with 1% of thiourea during 18 days. Follicular lumen reduced to a narrow cleft because of the reabsorption of the colloid. Colloid droplets in the cells.

10 Thyroid gland of a rat treated with thiouracil for 162 days. Follicles have regular amount of colloid, cells are higher than in the normal but much lower than in figures 8 and 9, and have also colloid droplets; connective tissue little increased.

11 Thyroid gland of a rat treated with 1% of thiourea during 13 days. Both follicles have several macrophages in the lumen.

12 Gland treated as in figure 11. Arrows indicate the basal colloid.

ON THE "CONICAL OPENINGS" IN THE WALL OF VENOUS SINUSOIDS AND THEIR RELATION TO THE SO-CALLED ERYTHROGENIC CAPILLARIES IN THE BONE MARROW OF MAN

EMIL MARO SCHLEICHER[1]
Department of Internal Medicine, Minneapolis General Hospital, Minnesota

ONE PLATE (TWO FIGURES)

The introduction of intrasternal-intratibial infusion and aspiration methods in the field of medicine renewed interest in the blood vascular system of the bone marrow, particularly in the "conical openings" in the wall of venous sinusoids which, according to a prevalent view, bear directly on the problem of erythropoiesis and delivery of erythrocytes in the general circulation.

Neumann (1869) described a closed "Capillarnetz" with "divertikelartige Ausstülpungen" in the bone marrow of the rabbit and thus established for the first time that the bone marrow is a vascular organ. Rindfleisch (1880) referred to this vascular plexus as "venöse Capillaren." Van der Stricht (1892) observed in bird and mammal "hematopoietic capillaries." In birds, these capillaries were closed to the general circulation. In mammals, on the contrary, the endothelial membrane of the "hematopoietic capillaries" was open, permitting the peripheral blood to circulate freely within the meshes of the reticulum. Doan ('22a, '22b, '25) produced a hypoplastic state of the bone marrow in the pigeon and injected at a pressure of 130 mm Hg a mixture of one part of India ink and three parts of physiological sodium chloride solution.

---

[1] Parke Davis Fellow in Clinical Hematology.

His study material consisted of cleared gross specimens and celloidin sections 100 to 150 micra thick. Doan observed between patent sinusoids and aggregated fat cells "collapsed capillaries" which he designated "intersinusoidal capillaries." These vessels terminated as "conical openings" leading into patent sinusoids. He advanced the point of view that these "intersinusoidal capillaries" are lined by hematogenic endothelial cells which in birds as well as in men proliferate "megaloblasts" (early pronormoblasts of other authors). Drinker and associates ('22) perfused mammalian bone marrow with hirudinized blood and physiological sodium chloride solution. These investigators could not demonstrate openings in the wall of the venous sinusoids and thus formulated the hypothesis that the extremely delicate endothelial walls of the capillaries are grown through by red cells in varying stages of maturity. Peabody ('26) observed funnel-shaped structures communicating with venous sinusoids in human femur marrow obtained at autopsy. According to this investigator the sides of the cones are formed by endothelium of the sinus, the upper border by the nucleus of an endothelial cell and the mouth or floor by the continuous endothelial wall of the sinusoid. He suggested that through these funnels mature erythrocytes enter the general circulation. Peabody based this opinion on the presence of erythrocytes within some cones. He conjectured that erosion of the floor membrane makes possible the entry of the red blood cells in the blood stream. Bargmann ('30) studied the blood vascular system in the bone marrow of the frog, rabbit, cat and dog. He observed neither erythrogenesis within "intersinusoidal capillaries," nor was he able to demonstrate conical openings communicating with sinusoids. He believes that the walls of the sinusoids are intact and not in continuity with the reticulum. It is Bargmann's view that the erythrocytes are released in the circulation by periodic "Durchbrechungen der histiocytären Wandmembran." Gohs ('34), who investigated the vascular plexus in the bone marrow in chicken, suggests that venous sinusoids are lined by endothelial cells morphologically difficult to separate from reticulum cells. He observed active hematopoiesis within sinusoids and is of the opinion that the latter connect directly with the venous system. Ringoen ('35) claims that, in birds, "stromal spaces" open into venous sinusoids. According to his point of view, the stromal spaces are lined by histiocytic elements which exhibit reticular fibers. Sabin and Miller ('38) show an 'erythrogenic capillary" opening into a sinusoid (femur marrow of a rabbit). The illustration also shows "normoblasts entering sinus at diverticulum of Neuman.[2] Jordan and Baker ('27) presume that the "endothelium stops when the venous sinusoidal system is formed." These investigators suppose that mesenchymal rather than true endothelial cells line the venous sinusoids. Corner ('20) does not favor this concept. He maintains that the capillary endothelium may become specialized and thus can produce reticulum fibers. Downey ('15) believes that cells which produce reticulum fibers are not endothelial cells.

The hypothesis that in man endothelial cells line the venous sinusoids and that these cells produce the fibers to which they are attached has certain points in its favor. It is a well known fact that capillary endothelium performs many functions. According to Zweifach ('40) the capillary endothelium acts as an "ultrafilter" permitting certain colloidal constituents of the blood to leak through the endothelial membrane. This selective filtration is determined by the degree of permeability or porosity and position of an endothelial membrane. There is reason to believe that the endothelial cells lining the venous sinusoids in the bone marrow in man perform similar if not identical functions. Physiologists and biochemists agree that diffusion of substances essential for the activation of resting reticulum cells and parenchymal growth in general occurs through the endothelial wall. The membranous barrier acts not only as a selective filter system but also permits cellular elements that have reached a certain physico-chemical state of their colloid envelopes to pass through the sinusoidal wall leaving no trace of their passage. Thus a mechanism (state of permeability) is brought into play that is similar if not identical to that which allows extravasation of erythrocytes from capillary endothelium, as for example the formation of petechia. Rohr ('45) stresses that the capillary network, particularly the sinusoids, has a "lückenlos geschlossenes Gefässendothel" and suggests that the entry of erythrocytes in the general circulation may come about by a "Wachstumsbruch unter temporärer Kontinuitätstrennung der Kapillarwände."

---

[2] The diverticulum of Neumann is not a normal anatomical structure. It is caused by unequal pressures on either side of an exceptionally thin sinusoidal wall which may, therefore, bulge outward or inward. Such a weakened wall can be easily injured by infusates or post-morten lysis. Rupture may occur creating an opening in the wall which obviously can be subject to misinterpretation.

The above brief review of opinions with respect to the presence or absence of "conical openings" and, by the same token, of erythrogenic capillaries, and whether the cones are open or closed to the general circulation shows clearly that the subject is of a highly controversial nature. Re-study of the problem was, therefore, believed worthwhile.

It is the purpose of this paper to record (a) that in the bone marrow in man conical structures are present which are formed when "intraparenchymal sinuses" are created by loss of fluid from fat cells, (b) that the sides of the cones are formed by fat cells rather than capillary endothelium, the top, when present, by a reticulum cell and the floor by the continuous wall of venous sinusoids, and (c) that the cones do not communicate directly with venous sinusoids, but open into peripheral sinuses (reservoirs).

METHODS AND MATERIAL

The methods employed were those of Schleicher ('44, '46), Semsch and Stoesser ('45) and Tucker ('45). The bone marrow tissue was obtained from the sternum at the level of the second and third costal interspace of a young woman who received 1000 cm^3 of fresh blood plasma by the intrasternal route for therapeutic purposes. The plasma was administered by the continuous drip method and through a standard sternum needle [3] which was introduced in the medullary cavity at the level of the second intercostal space. The flow of the blood plasma is regulated by noting the appearance of a "sharp pain" about the infusion area experienced when the pressure of the incoming fluid is greater than the intramedullary pressure. In this particular case the blood plasma was fed at a rate of 80 drops per minute. About 3 cm^3 of bone marrow was obtained before and near the completion of the infusion.

The first specimen served as a control. The second sample was aspirated at the level of the third intercostal space to prevent aspiration of injured tissue from the first sampling. Microscopic examination of serial sections (5 micra thick) of the control specimen revealed an occasional "conical structure" adjacent to venous sinusoids. The latter were few and mostly collapsed, and the cones were difficult to make out even in areas where fat cells bordered venous sinusoids. In the infused marrow, however, the majority of venous sinusoids were dilated and conical structures contiguous to the sinusoidal wall were clearly visible. Most of these funnel-shaped structures were empty. When cellular elements were present the cell mass was only occasionally of one type of cell. The most frequently encountered cellular content was made up of erythrocytes, few basophilic and orthochromatic normoblasts, some polymorphonuclears and thrombocytes. A few cones contained metamyelocytes and polymorphonuclears only, and again others had, besides myeloid cells, erythrocytes and small elements interpreted to be thrombocytes admixed. In areas which showed fatty metamorphosis, conical structures were present between each pair or between pairs of fat cells. An example of the type of conical structures observed in the infused marrow is shown in figure 1 and described in detail in the legend. Empty cones varied in hue from colorless to light reddish-blue with an occasionally more deeply stained area just below the top reticulum cell. This homogenous-to-granular mass was presumed to be concentrated protein-lipoid substances derived from the infused blood plasma. In some of the serial sections the wall of the venous sinusoids appeared to be fused to the surrounding parenchyma, giving the impression that the conical structures opened directly into the venous sinusoids. Serial sections stained with silver impregnation methods ('27, '41, '46) and connective tissue stains ('43) revealed that the cones opened into ''peripheral sinuses'' rather than venous sinuosids. Furthermore, these special preparations re-confirmed the observation made on hematoxylin-and-eosin stained sections that the conical structures were part of ''intraparenchymal sinuses'' (presumed to be created by loss of fluid from fat cells) rather than the terminus of so-called ''erythrogenic capillaries.'' The existence of the latter could not be verified in the material studied. The ''intraparenchymal sinuses'' communicate with hematopoietic islets, that is, erythrogenic, myelogenic and megokaryocytogenic areas. These sinuses were either single, or fork-like, or formed interlacing channels resembling a network. They varied in length, width and tortuosity. A more or less well formed conical structure marked the point of union between an ''intraparenchymal sinus'' and ''peripheral sinus.'' A ''Y'' shaped ''intraparenchymal sinus terminating in a ''peripheral sinus'' is shown in figure 2 and described in detail in the legend. The peripheral sinuses varied in length, width and cellular content. A peripheral sinus, one cell large in diameter, is shown in figure 1, inset. To follow the course of such a narrow sinus when empty, may prove difficult indeed, particularly when the sinusoidal wall (outer boundary) is closely attached to the surrounding tissue. In hyperplastic areas where shrinkage and disappearance of fat cells created ''cell pools'' of various dimensions the identification of intraparenchymal sinuses, conical structures and peripheral sinuses becomes quite arduous.

---

[3] Made by V. Mueller and Co., 408 S. Honore St., Chicago, Ill.

Based on the study reported here, the following concept may be formulated. ''Intraparenchymal sinuses'' permit migration of non-motile and motile cellular elements toward the wall of venous sinusoids. The latter are part of the general circulation. The mouth of the intraparenchymal sinus has a conical shape which also marks the beginning of the "peripheral sinus" (reservoir). When the cones and peripheral sinuses are empty, migration and delivery of cellular elements has occurred. Small islets of immature cells may gravitate into the cone and become lodged within the borders of the peripheral sinus. Such gravitated islets may cause bulging of an exceptionally thin sinusoidal wall forming the so-called "diverticulum of Neumann." It appears that under physiological conditions that next to mature cells there are also immature elements of various developmental stages present in the peripheral sinuses. It is hypothetically possible that the reservoirs offer a more favorable environment for cellular metabolism. It is obvious that filtration or diffusion of growth promoting substances presupposes a "gradient," that is, a physico-chemically altered wall of the venous sinusoids and by the same token specific stimuli can quickly mobilize cellular elements which have reached maturity or a certain state of development of their colloid envelopes and thus can traverse the membranous wall leaving no trace of their passage.

SUMMARY

1. In non-infused and infused serially sectioned human sternal bone marrow obtained during life, "conical openings" in the wall of venous sinusoids connecting so-called erythrogenic capillaries with the general circulation were not observed.

2. "Conical structures" were noted which marked the union between "intraparenchymal sinuses" and "peripheral sinuses."

3. Fat cells formed the sides of the conical structure, the top, when present, was formed by a reticulum cell, and the floor or mouth by the continuous endothelial wall of the venous sinusoid.

4. It could be demonstrated that, besides erythroid cells, myeloid elements and thrombocytes were present in various quantities within the conical structures.

5. The conical structures are formed when fluid escapes from fat cells, creating "intraparenchymal sinuses" (channels). By means of these channels cellular elements move toward the continuous wall of venous sinusoids. The latter are part of the capillary plexus. The sinusoidal wall acts as a barrier between the peripheral sinuses and the general circulation.

6. The bone marrow was obtained from the sternum at the level of the second and third costal interspace. A standard infusion and aspiration method and a standard sternum needle were employed. The infusate was fresh human blood plasma.

7. The prevalent view that erythrogenic capillaries exist in the bone marrow in man, and that conical openings connect them with venous sinusoids and indirectly with the general circulation, and that these cones bear directly on the problem of erythropoiesis and delivery of erythrocytes in the peripheral blood stream could not be verified.

LITERATURE CITED

BARGMANN, W. 1930 Über den Feinbau der Knochenmarkskapillaren. Zeitschr. f. Zellforsch. u. Mikr. Anat., vol 11, p. 1.

CORNER, G. W. 1920 On the widespread occurrence of reticular fibrils produced by capillary endothelium. Carnegie Institution Wash. Contrib. Embryol., vol. 9, p. 85.

DOAN, C. A. 1922a The capillaries of the bone marrow of the adult pigeon. Johns Hopkins Hosp. Bull., vol. 33, p. 222.

———— 1922b The circulation of the bone marrow. Carnegie Institution Wash., Publ. 67. Contrib. to Embryol., vol. 14, p. 27.

DOAN, C. A., R. S. CUNNINGHAM AND F. R. SABIN 1925 Experimental studies on the origin and maturation of avian and mammalian blood cells. Carnegie Institution, Wash., vol. 16, p. 163.

DOWNEY, H. 1915 The so-called "endothelioid" cells. Anat. Rec., vol. 9, p. 73.

DRINKER, C. K., K. R. DRINKER AND C. C. LUND 1922 The circulation of the mammalian bone marrow. Am. J. Physiol., vol. 62, p. 1.

DUBLIN, W. B. 1946 Reticulum. Arch. Path., vol. 41, p. 299.

GOHS, W. 1934 Zur Frage der Blut- und Gefässbildung im Knochenmark. Virch. Arch., vol. 294, p. 103.

JORDAN, H. E., AND J. P. BAKER 1927 The character of the wall of the smaller blood vessels in the bone marrow of the frog with special reference to the question of erythrocyte origin. Anat. Rec., vol. 35, p. 161.

NEGRIN, J. 1941 A silver impregnation method for reticulum which may be used with any fixative and any embedding medium. Arch. Path., vol. 31, p. 108.

NEUMANN, E. 1869 Ueber die Bedeutung des Knochenmarkes für die Blutbildung. Arch. d. Heilk., vol. 10, p. 68.

ORSOS, F. 1927 Das Bindegewebsgerüst des Knochenmarks in normalen und pathologischem Zustand. Beitr. path. Anat., vol. 76, p. 33.

PEABODY, F. W. 1926 The study of the hyperplasia of the bone marrow in man. Am. J. Path., vol. 2, p. 487.

RINDFLEISCH, G. E. 1880 Ueber Knochenmark und Blutbildung. Arch. Mikr. Anat., vol. 17, p. 21.

RINGOEN, A. R. 1935 The so-called "Intersinusoidal Capillaries" of avian bone marrow. Anat. Rec., vol. 61 (suppl.), p. 42.

ROHR, K. 1945 Funktionelle Knochenmarkspathologie. Schweiz. Med. Wochenschr., vol. 75, p. 773.

SABIN, F. R., AND F. R. MILLER 1938 Normal Bone Marrow. Downey's Handbook of Hematology, New York, Paul B. Hoeber, Inc., vol. 3, p. 1815.

SCHLEICHER, E. M. 1943 Mallory-Heidenhain's connective tissue stain employing domestic azurcarmin B, aniline blue and orange G in balanced proportions. Am. J. Clin. Path., vol. 4, p. 35.

——— 1944 The volumetric pattern of aspirated normal human sternal marrow of males 18 to 40 years. Am. J. Clin. Path., vol. 14, p. 1011.

——— 1946 Miliary tuberculosis of the bone marrow. Am. Rev. Tubercul., vol. 53, p. 115.

SEMSCH, R. D., AND A. V. STOESSER 1945 Intratibial infusion in children. J. Lancet, vol. 65, p. 194.

TUCKER, B. 1945 A method for histologic preparations from gross marrow units. Minnesota Med. Technol., vol. 8, no. 3.

VAN DER STRICHT, O. 1892 Nouvelles recherches sur la genése des globules rouges et des globules blancs du sang. Arch. de Biol., vol. 12, p. 199.

ZWEIFACH, B. W. 1940 The structural basis of permeability and other functions of blood capillaries. Cold Spring Harbor Symposia Quant. Biol., vol. 3, p. 216.

PLATE 1

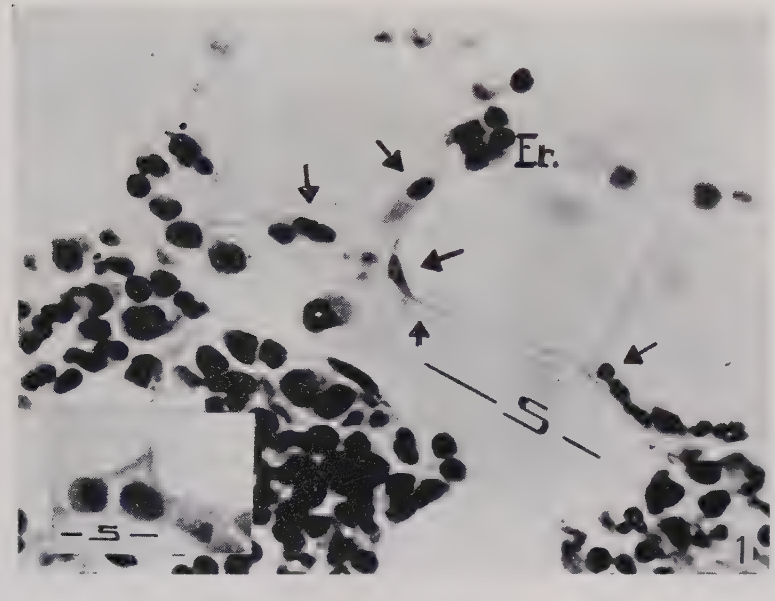
EXPLANATION OF FIGURES

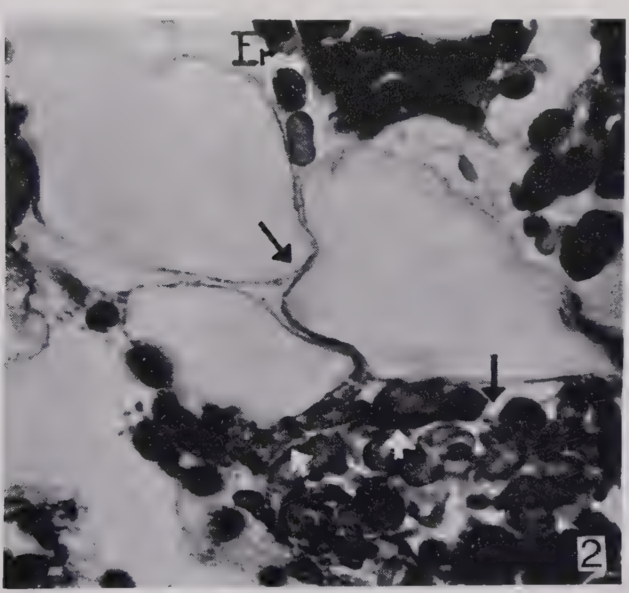
Photomicrographs of infused sternal bone marrow. Serial sections about 5 micra thick. (Photomicrographs by Mr. H. W. Morris, Medical Photographer, University of Minnesota.)

1 Letter ''S'' identifies a venous sinusoid. A small hematopoietic area (erythroblast isle) at letters ''Er.'' Directly below the isle is a ''conical structure.'' The upper arrow points at the top of the cone which is formed by a reticulum cell (presumed not to be activated), the sides are formed by fat cells. The lower left arrow points at an oval nucleus of the continuous wall of the venous sinusoid which forms the floor of the cone. The lower right arrow identifies the nucleus of one of the lining fat cells. The small round structure at the floor of the cone is an erythrocyte out of focus. Protruding into the lumen of the venous sinusoid are several small bodies believed to be thrombocytes.

The arrow in the lumen of the venous sinusoid points at an empty portion of a faintly stained ''peripheral sinus'' and the extreme right arrow points at erythroid and myeloid cells within the same sinus. Near the extreme right arrow a compressed ''intraparenchymal sinus'' coursing between fat cells toward the upper right corner of the photomicrograph. The sinus contained material which had the consistency and color of coagulated plasma.

2 The upper black arrow identifies a winding ''intraparenchymal sinus'' originating at point ''Er.'' (erythroblast isle). A late basophilic normoblast and two erythrocytes are at the place of origin of the sinus. Near the arrow the sinus is joined by another ''intraparenchymal sinus'' which communicates with a myeloid isle at the extreme left. The common sinus terminates into a ''peripheral sinus'' which is identified by the lower black arrow. Fat cells (note degree of distortion) form the sides of the ''intraparenchymal sinuses'' and the floor of the peripheral sinus is formed by the wall of a venous sinusoid which is identified by the two white arrows within the lumen of the vessel. The peripheral sinus and the sinusoid contain erythroid and myeloid cells. The white arrow on the right points at an elongated nucleus of the wall of the venous sinusoid and the white arrow at the left points at a swollen part of the sinusoidal wall. This structural alteration of the sinusoidal wall is quite striking when compared with the membranous lining of slightly dilated venous sinusoids in the control sternal marrow (fig. 1, inset).


Er.
S
S
1


Er.
2

EMBRYONIC AND POSTNATAL DEVELOPMENT OF THE ADRENAL CORTEX, PARTICULARLY THE ZONA GLOMERULOSA AND ACCESSORY NODULES

PETER GRUENWALD[1]
Department of Pathology, Children's Hospital and Infants' Hospital,
Boston, Massachusetts

THREE PLATES (EIGHTEEN FIGURES)

Recent investigations of the adrenal have suggested that new cortical tissue differentiates in the capsule of the mature gland, and subsequently becomes part of the zona glomerulosa (Zwemer, Wotton and Norkus, '38; Bachmann, '39; Gruenwald, '42a; Wotton and Zwemer, '43; Gruenwald and Konikov, '44). A close relationship of the cells of the capsule to those of the cortex is further suggested by the histological demonstration of ascorbic acid (Heckel, '42) and of vitamin A (by fluorescence microscopy, Popper, '41) in the capsule. The mechanism of incorporation of newly formed cortical tissue into the cortex proper depends on its amount, and on the structure of the zona glomerulosa (Gruenwald and Konikov). It has also been claimed, that in this region, as well as at the inner border of the cortex, not only differentiation but also degeneration of cortical tissue may occur under the control of the corticotropic hormone of the hypophysis (Tonutti, '41, '42b). Thus, the examination of the capsule and zona glomerulosa may, after elucidation of the normal phases of their changes, become an important part of the study of the adrenal cortex under experimental and pathological conditions.

---

[1] Author's present address: Department of Pathology, Long Island College of Medicine, Brooklyn, N. Y.

In the course of an examination of large numbers of human adrenals it became apparent that the structure of the zona glomerulosa, and the form and amount of apparently newly formed cortical tissue, show considerable changes during infancy and childhood. The present work is concerned mainly with this aspect of cortical development. The structure of the outermost layers of the human adrenal and its changes from embryonic life to maturity will be reported, including findings in accessory cortical nodules. This work is limited to observations made with the usual histological methods applied to paraffin sections. Only in a few instances was it supplemented by the examination of sudanophil lipoid in frozen sections. No studies were made of evidence of endocrine and other specific metabolic activity of the cells by physical or histochemical methods, such as the demonstration of steroids, plasmal, or vitamins A or C. The present results are intended to provide a basis for further histochemical, experimental and pathological work, and are not expected to be final until confirmed by these methods.

The following excerpts from the literature on adrenal development will serve as a starting point for the discussion of the observations. Many authors have examined the early development of the organ in man and mammals from the coelomic wall, and most of them have come to the conclusion that epithelial cords of the cortex primordium arise as outgrowth of the coelomic epithelium. A recent representative of this opinion, reporting on human embryos, is Politzer ('36). Goormaghtigh ('21) and Waring ('35) failed to find distinct epithelial buds, and more recently Uotila ('40) said of the cortex primordia in human embryos that "the formation of distinct cell buds has not been observed." In a previous investigation, the present author ('42a) also failed to find epithelial cords forming the early cortex primordium. The cell mass from which the cortex arises is purely mesenchymal in its structure for a considerable time after its formation, as can easily be demonstrated by impregnation of lattice fibers surrounding each cell. The cells are part of the mesenchyme of the coelomic wall, and many of them derive from dividing cells of the coelomic lining in much the same way as do many cells of the nearby connective tissue. The lining of the coelom is not epithelial itself at this time and place, and when it gives rise to cells of the cortex anlage, it does so without the formation of epithelial cords. It was also pointed out that, judging from the presence of lattice fibers enveloping single cells, the greater part of the adrenal cortex permanently remains non-epithelial. Only a peripheral layer, roughly corresponding with the zona glomerulosa, gradually loses the argyrophil fibers within its cell cords (Gruenwald and Konikow), and is then truly epithelial. Most adrenals show fluent transitions between epithelial and non-epithelial structure at the border of the two portions of the cortex.

Much has been written about that part of the adrenal cortex of the human fetus which degenerates shortly after birth, and is usually referred to as fetal cortex or x-zone. The former name is not entirely correct because the adrenal cortex of the fetus contains the permanent as well as the degenerating portion. In view of our incomplete knowledge of the nature of the latter portion, the term x-zone appears most appropriate. Time and manner of its degeneration have been thoroughly studied by Lewis and Pappenheimer ('16) and Benner ('40). Only a few mammals have an x-zone, among them the mouse, and in this species differences were found between various strains. The x-zone of the mouse has been subjected to experimentation, and from its reaction to hormonal influences Gersh and Grollman ('39) concluded that it is not essentially different from the rest of the cortex.

Age differences in the postnatal human zona glomerulosa have, to the best of the author's knowledge, not been investigated. Gruenwald and Konikov briefly described such differences in cats.

Many authors assume that there is a cell movement in the mature adrenal cortex from the periphery toward the zona reticularis. The literature on this subject may be found in the articles of Blumenthal ('40) on mitotic activity in the outer part of the zona fasciculata, and of Hoerr ('31) and Bennett ('40) dealing with the changing properties of cortical cells in various layers. Cell movement from the site of mitotic activity in the outer zona fasciculata toward the zona reticularis obviously does not concern the zona glomerulosa, and many authors have failed to consider the role of the latter zone in the life and function of the adrenal cortex. Mulon ('03) regarded the zona glomerulosa as the germinal layer, but his findings of frequent mitosis and amitosis in that zone have not been confirmed. Others ascribe to the zona glomerulosa the role of a cell reservoir. This has been discussed and older literature quoted by da Costa ('22, '33). A tentative concept, including this phase as well as the more recently discovered new formation of cortical tissue in the capsule, has been proposed by Gruenwald and Konikov ('44). According to this hypothesis, multiplication of cortical cells by mitosis in the outer fasciculata may be supplemented or replaced by the release of cells from the reserve store in the zona glomerulosa. Depleted stores may in turn be replenished either by mitosis in the outer fasciculata or by apposition of newly differentiated cortical cells from the capsule. In support of the reservoir function of the zona glomerulosa, its transformation into part of the zona fasciculata has been described (Goormaghtigh, '18; da Costa, '33; Gruenwald and Konikov, '44), apparently under the influence of a demand for functional cells.

Several authors do not agree with this concept of inward movement of cells in the mature adrenal cortex. Whitehead ('43) claims that mitosis occurs in the cortex solely during the growth period and not in adult animals, and that only growth of the cortex, but not continuous cell replacement, occurs by cell division. Blumenthal ('45) recently found a decrease, but no complete disappearance of mitoses even in old guinea pigs. Tonutti ('41, '42) holds that growth as well as degeneration occurs simultaneously at the outer and inner border of the cortex, and that there is no cell movement toward the zona reticularis.

MATERIAL AND METHODS

The early development of the adrenal cortex in embryos of the second month has been described in a previous report ('42a). The same material, consisting of thirty-seven serially sectioned human embryos of 7.5 to 32 mm, was again reviewed, mainly in order to examine the alleged separate origin of the permanent cortex by a second proliferation from the coelomic lining (Keene and Hewer, '27; Uotila, '40). The sections of these embryos are stained mostly by the azan method; in a smaller number of specimens impregnation of lattice fibers (method of Gomori) was used. Adrenals of ten human embryos of 6.7 to 22 cm crown-heel length were also available. In each case at least part of one organ was sectioned serially and various stains were employed, among them in all cases, the azan method and silver impregnation.

Postnatal material was selected from a large number of routine sections of organs fixed in Zenker's fluid, and new sections of suitable specimens were cut and stained by various methods. The age distribution of the postnatal material is as follows:

Newborn to 1 day	8
1 day to 1 month	12
1 to 6 months	19
6 to 12 months	8
1 to 2 years	10
2 to 3 years	8
3 to 10 years	15
10 to 20 years	9
20 to 78 years	24

Organs of several premature infants are included in this material.

Selection of proper staining methods proved to be of great importance in the present work. The usual hematoxylin and eosin stain does not show adequately the form and arrangement of the cortical cell groups. It is necessary to stain the connective tissue fibers which bind these cell groups. Stains for collagenous fibers are inadequate, particularly in organs of embryos and infants which are devoid of collagenous fibers, except in the densest portion of the capsule and the walls of larger blood vessels. Impregnation of lattice fibers gives an excellent view of the architecture of the adrenal cortex, even in those cases in which many other stains show but a uniform mass of cells, such as in the outer portion of the fetal adrenal. In addition, other stains were used to show the cells themselves. A combination of any of these stains with silver impregnation to the extent of showing much cellular detail has not been used because fine lattice fibers are then obscured.

OBSERVATIONS

Embryos of the second month. Previous work on the origin of the adrenal cortex primordium was reviewed above, including the author's own findings favoring the mesenchymal origin of the anlage ('42a). According to Keene and Hewer ('27) and Uotila ('40), a second proliferation of the coelomic lining takes place in embryos of about 12 mm, and the cells thus formed (the epithelial cap of Keene and Hewer) surround the first anlage and form the permanent cortex. Examination of embryos at this age shows, particularly with the use of silver impregnation, that the adrenal primordium contains no epithelial cell groups. The cortical cells are swollen cells of the mesenchyme, and it is difficult if not impossible to distinguish the superficial cortical cells from the adjacent mesenchyme at all points. Proliferation of the coelomic lining still occurs at the stage designated by the above mentioned authors but, just as in the earlier phase, not in the form of epithelial sprouts. Instead, more mesenchyme is formed in the adrenal region, and it is probable that some of the cells partially surround the original anlage and then differentiate into cortical cells. It is difficult to obtain reliable information on the amount of cortical tissue thus formed, and the impression just described was obtained by judging the distance of the cortex primordium from nearby structures and thus the growth of the organ at the expense of the mesenchyme. Nothing was seen to confirm the alleged migration of cells derived from the coelomic lining around the entire cortex primordium.

These observations lead to the conclusion that the adrenal cortex develops by rapid differentiation of mesenchyme of the coelomic wall during the early phase and a slow continuation during a subsequent phase, rather than a succession of two separate proliferations. No epithelial cell groups are present in the primordia during this and a long successive period (fig. 1).

When proliferation of mesenchyme from the coelomic lining and differentiation of cortical cells from the mesenchyme cease, the adrenal is composed of swollen cells which are large and have a more acidophilic cytoplasm in the central portions than in the periphery. Each cell is surrounded by a network of lattice fibers. Wide blood vessels make their appearance in the central part of the organ and give it a spongy structure while the distal zone remains solid (fig. 1). Gradual transitions lead from large to small cells, and from spongy to solid arrangement. It is impossible at any stage of intrauterine life to draw a sharp line between cells which differentiated at different times in the early embryo, or between x-zone and permanent cortex, or any other layers in the cortex. Just as in the mature organ, gradual transitions are seen everywhere, due perhaps to gradual changes in the differentiation of cells traversing the zones. It is, therefore, unjustified to speak of separate anlagen of the x-zone and the permanent cortex.

Later embryonic stages. In the course of several months of embryonic life, two zones joined by an area of transition develop in the adrenal cortex. (The temporary presence in the cortex of groups of migrating cells of the future medulla will not be described.) Lattice fibers separating each cell from its neighbor disappear in the outer zone of small cells (fig. 2; for an early stage of this change in the dog embryo see Gruenwald and Konikov, fig. 23). The difference in cell size between the inner and outer zone becomes more marked (fig. 3). As a result, the outer zone consists of large, solid, epithelial cell masses, subdivided by blood vessels and lattice fiber trabeculae at a great distance from one another. On section, the width of these cell masses from one connective tissue partition to the next varies considerably. In some areas no such partitions are present throughout an entire low-power field or a large part of one, while in other regions many more subdivisions may be found. There seems to be a trend toward division of larger cell complexes in the outer zone into smaller ones during late embryonic life.

This outer layer of the embryonic cortex has been considered either as the zona glomerulosa or as the permanent cortex, in which case it would correspond to both the zona glomerulosa and the permanent zona fasciculata of the postnatal gland. It was pointed out above that the existence of fluent transitions between zones makes any comparison of layers at different stages very unreliable. When the x-zone degenerates after birth some of the large cells always remain intact and eventually become part of the permanent zona fasciculata. Comparative considerations are also in favor of the origin of the permanent zona fasciculata from the large cells of the embryonic gland because these cells are also present in the embryos of those species which have no x-zone that degenerates after birth.

The well known cavities appear in the outer zone of the embryonic adrenal shortly after it has acquired its epithelial structure. Their size and number vary greatly. Figure 3 shows an example of an organ with numerous cavities. Their morphogenetic importance is not understood. We do not know whether their appearance is an essential part of cortex development, or an unimportant event. Hett ('25) favors the first mentioned view, and believes that the formation of the zona glomerulosa with its arches and cell balls depends on these cavities. Many other workers have described them without attributing to them much importance for the future development of the gland. Da Costa ('28) sees in the cavities an expression of a fundamental glandular structure of the cortical cell cords. In human embryos the cavities show considerable variation in number and are present for a varying length of time. In some cases they persist after birth for many months (fig. 8), and similar cavities have been found in adult mammals (Kolmer, '18; Kohno, '25; Da Costa, '28). Formation of the zona glomerulosa, on the other hand, proceeds in human embryos simultaneously all over the surface of the organ. This, as well as the presence of cavities long after differentiation of the zona glomerulosa, speaks against Hett's assumption of an important role of these spaces in the development of the zona. As has just been described, the zona glomerulosa develops by a slow process of differentiation characterized by the disappearance of lattice fibers surrounding single cells, the appearance of a characteristic difference of cell size and staining properties between these cells and those of the deeper zona, and the formation of large cell masses connecting with many of the slender cell cords of the deep layer.

Postnatal development. It is difficult to describe definite stages in the postnatal development of the adrenal cortex because of a wide variation not only in the time at which certain changes occur, but also in the stages of development found in adjacent regions of the same gland. The only change in the infant's adrenal which shows a fairly close correlation with age is the degeneration of the x-zone, and this will not be described here. In presenting descriptions and photographs of parts of adrenals, it will always be understood (unless otherwise indicated) that the condition is typical of the given stage but not the only one to be seen in that organ, and that it may occur occasionally in younger glands and frequently in older ones.

Newborn to 6 months. The adrenal cortex of the newborn consists of the two layers which were described above. The greater part of the inner layer of large cells forms the x-zone and degenerates during the first few months of extrauterine life. Its demarcation from the permanent cortex is not a sharp one until the process is far advanced; the difficulties in establishing the identity of the x-zone with any part of the fetal organ have been referred to above.

At birth the zona glomerulosa consists of the wide arches found in the fetus. It is difficult to visualize the shape of these units even with the aid of serial sections. They are probably irregular plates extending parallel to the capsule and connecting on their inner side with the ends of large numbers of fasciculata cords (fig. 4). Persistence of wide undivided glomerulosa units resembling those of the fetus was seen in a 2½ months old infant in an otherwise normal adrenal (fig. 6). The mechanism of subdivision of large units into smaller ones is not apparent from the study of the present material. In one instance the appearance of a lattice fiber framework within large glomerulosa units suggests a change of differentiation and arrangement (fig. 5), producing narrow glomerulosa cords, while heavier fibers still indicate the boundaries of the old units.

There is also great variability in the height of the glomerulosa units from the capsule to the level at which lattice fibers appear between the cells (compare figs. 4 and 6). In young infants the zona glomerulosa consists of small cells with dark staining nuclei as described above in fetal glands. There is a gradual transition to larger cells with a very pale staining cytoplasm and somewhat larger nuclei, giving the entire layer a pale appearance when stained with the usual methods. This change may occur at any time between the ages of 2 weeks and 6 months. It is preceded by a gradual decrease in width of the zone of small cells, and does not affect all cell groups simultaneously so that dark and pale staining glomerulosa units may be present side by side (fig. 7). Sudan-stained frozen sections show numerous small lipoid droplets in the pale staining glomerulosa units. The change just described is preceded by the disappearance of the cavities which are present in varying numbers of glomerulosa units of younger organs. The oldest gland in which numerous cavities were seen, at the age of 6 months, is shown in figure 8. In this case, however, debris is present in many of the cavities, suggesting that they may not be strictly comparable with fetal cavities.

Accessory cortical nodules in the vicinity of the main gland are found very frequently in infants. According to the review of Dietrich and Siegmund ('26) they are present in almost all individuals during the first year and decrease in number during childhood. In the present material, adrenals of thirty-four infants up to 3 months of age were examined in single routine sections, and twenty-four of these sections showed accessory nodules. In most cases multiple cell groups were found in or near the capsule. It is probable that most of the remaining ten cases also had accessory nodules which were not cut in these single sections. Figure 10 shows the case in which the largest number of accessory cortical cell groups was present.

Accessory nodules appear in various forms and varying relations to the main gland. Often they are spherical and, if large enough, show the same zones as the main gland in corresponding stages of their development. This includes degeneration of the central portions of the nodules at the time when the x-zone degenerates in the adrenal proper. Many of these nodules contain in their own capsule cords or sheets of cortical cells (fig. 12) which closely resemble those present in the capsule of the main glands, only in later stages. That suggests that accessory nodules may grow by apposition of newly differentiated cords at a time when this is uncommon in the main gland. Thomas ('11) described this long before the discovery of differentiation of cortical cords in the capsule of mature adrenals. He assumed that cortical cells had split off from the primordium of the adrenal in the early embryo, and remained inactive in the capsule until some unknown stimulus caused their differentiation. Since we assume that cells of the capsule and nearby connective tissue have the potency to form cortical tissue, the assumption of cells splitting off from the embryonic adrenal has become superfluous. Accessory nodules far from the main gland develop like the ones in the capsule. Figure 13 shows a nodule in the epididymis of a 2 months old infant with cortical cords in the surrounding connective tissue and far advanced degeneration of the x-zone in the center.

Occasionally the capsule of the adrenal proper contains groups of cortical cell sheets resembling those in older children and adults, which are believed to replenish the cortex by apposition (Zwemer, Wotton and Norkus, '38; Wotton and Zwemer, '43; Gruenwald and Konikov, '44). However, these formations in young infants differ from those of older individuals by forming compact groups which reach a considerable size and differentiation before a narrow connecting bridge to the main gland is established (fig. 11). In this respect they resemble accessory nodules rather than stages of apposition. Single cords joining the cortex, as in accessory nodes or in the main glands of older persons, do not occur in the adrenals proper of young infants.

Accessory cortical cell masses in the capsule may be seen in all stages of fusion with the main gland. This may be interpreted as indicating either separation from the main gland, or independent origin and secondary fusion with it. Stecksén ('02) favored the former alternative. Against this is the decrease in the number of accessory nodules with advancing age (see above), and the fact that growth of accessory nodules (and in later stages also the main gland) by apposition can easily effect a fusion if it fills the space between the nodule and the adrenal proper. In those cases in which the accessory tissue is present in the form of cords and sheets of cortical cells spread between the connective tissue layers of the capsule, outgrowth from the main gland cannot very well be assumed; it is very doubtful that normal cortical tissue should be capable of this extent of infiltrative growth. Degeneration of cortical nodules by extreme vacuolation and fatty changes of their cells (Stecksén) has not been found in the present material. Perhaps lobules of immature fat tissue with foamy cells, which are often seen near the capsule of the adrenal in infants, were mistaken in the past for degenerating cortical tissue.

The old idea of the origin of accessory cortical nodules by deviation of epithelial sprouts in the early embryo was based on the assumption that the normal cortex itself is derived from epithelial cords growing in from the peritoneal epithelium. It was mentioned above that this is not the case. There is reason to believe that the potency to form cortical tissue is not limited to the cells which do so in the early embryo. Evidence of this is the differentiation of cortical cell cords in the capsule of the mature organ, as well as the recently discovered development of functionally adequate cortical tissue in the ovaries of ground squirrels (Groat, '43, '44) and mice (Hill, '46) after adrenalectomy. In view of these facts it appears most probable that accessory cortical tissue forms by differentiation in loco of cells of the connective tissue which are normally endowed with the necessary developmental potency (Gruenwald, '42b).

The accessory nodule undergoes a characteristic change in the course of what is believed to be its junction with the adrenal proper. It is incorporated into the structural pattern of the main gland in the sense that its cell cords show transitions from glomerulosa to fasciculata structure no longer in the direction of the center of the nodule, but toward the center of the main gland. Even if there is only a narrow connecting bridge, fasciculata cords traverse it and continue from the nodule through a breach in the zona glomerulosa directly into the fasciculata of the main gland.

One-half to 3 years. During this period the relative uniformity of the peripheral portion of the adrenal disappears. Areas of widely different structure may often be seen in one section. The transformation of the zona glomerulosa into a layer of pale staining cells with a foamy cytoplasm continues, and in many areas silver impregnation reveals a peculiar separation of this zone from the underlying parts by a continuous layer of lattice fibers (fig. 9). These fibers are obviously not derived from the capsule, and their presence under a superficial zone of cortical tissue cannot, as in the adult, be regarded as an indication that the outer zona has recently differentiated in the capsule. A similar separation of the zona glomerulosa from the fasciculata by lattice fibers has been found in adult cats, and in a striking form in cattle embryos (Gruenwald and Konikov, '44). Its significance is unknown. Frozen sections of the adrenal shown in figure 9 were stained with Scharlach R. The sharply delimited zona glomerulosa is very rich in lipoid, more so than the fasciculata.

In other parts of adrenals of this age group, apposition of newly formed cords has apparently begun or may begin at any time. These areas are found most commonly near the edge of the leaflets of the gland, and show thin cell cords in the zona glomerulosa which are not thicker than those of the zona fasciculata into which they continue proximally. Their distal ends are often thinned and curved in such a manner that they are almost or entirely parallel to the capsule. The ends of these cords can often be found between the fiber layers of the capsule (fig. 14). This condition is identical with that described as suggesting apposition of newly formed cortical cords (Zwemer, Wotton and Norkus, '38; Gruenwald and Konikov, '44).

In addition to the large and often proximally delimited glomerulosa units and the thin, oblique cords which have so far been described, the adrenals contain during the period under consideration many cell cords in their zona glomerulosa which are less conspicuous. These are mostly somewhat wider than fasciculata cords, and arranged in radial direction. As development goes on, these cords increase considerably in number, until they dominate the field in the adult.

The accessory nodules do not differ essentially from those described in the preceding age group. They correspond in their structure with the main gland if their size permits the differentiation of zones. They are often surrounded by large numbers of apparently newly formed cortical cords. One difference is often apparent between the adrenal proper and its accessory cell groups. The extensive accumulation of lipoid droplets in the cells of the zona glomerulosa is not duplicated in the accessory nodules. The glomerulosa cells of these nodules usually contain less lipoid than does their zona fasciculata.

Three to 10 years. All forms of glomerulosa units of the preceding age group may be seen in adrenals of children up to 10 years and older. In addition to these, a new form is comparatively frequent and often appears in large areas of the zona glomerulosa. Figure 15 shows a large portion of a leaflet from an adrenal of a 6-year-old child. Arches of cortical tissue resembling the zona glomerulosa of younger children are seen here not on the surface of the gland, but separated from the capsule by a conspicuous layer of thin cortical cords in an irregular arrangement. In some portions the superficial layer is thin, and the cords in it are arranged roughly parallel to the capsule (fig. 16). This suggests that the entire peripheral layer consists of newly differentiated cords laid down over the old zona glomerulosa. The other alternative, namely, outgrowth of these cords from the zona glomerulosa or development by transformation of the latter, does not fully explain the existence of wide and apparently undisturbed units of the zona glomerulosa in the depth. The relative frequency with which this condition is found indicates that it either persists with little change for a long period of time or occurs repeatedly.

In addition to accessory nodules similar to those previously described, a new form of cortical cell masses may now be seen (fig. 17). These are areas within the cortex of the main gland which are limited against the surrounding tissue either completely or only distally and on their sides. The peripheral cell groups in these regions have the character of zona glomerulosa even though they may be located at the level of the zona fasciculata. In their arrangement they appear to be part of a separate nodule. An explanation of the origin of these nodules is suggested by the fact that they were not seen in infants, and by the appearance of some of them in serial sections. They may then be seen to emerge on the surface of the organ and have the typical appearance of accessory nodules fused with the main gland. All this suggests that they are accessory nodules which have been partially taken up into the main gland. However, only a small proportion of the accessory nodules retain this individuality after their fusion with the adrenal proper.

Ten years through maturity. Histological evidence of apposition of newly differentiated cortical tissue from the capsule of the mature adrenal has been quoted above. The present description is intended merely to establish a link between the preceding sections and the better known condition in the adult. Whatever changes occur in the outer zone of the adrenal after the tenth year can no longer be broken down into stages associated with limited age periods because the great variability makes such classification impossible. The changes described in the preceding sections, such as division of large glomerulosa units into smaller ones, the appearance of cords radiating obliquely into the capsule, and the formation of new layers of cortical cords over the old zona glomerulosa, all tend to produce a glomerulosa consisting of thin cell cords. These form direct continuations of the fasciculata cords without a conspicuous change in caliber. Wide cell columns in the zona glomerulosa are relatively uncommon in mature adrenals. In some areas many thin cords radiate toward the capsule at various angles, joining apparently newly formed cords or offering opportunities for such junctions. In some instances these groups of cords have the shape of a wedge with the base at the capsule (fig. 18, left half and extreme right), and other groups of cords with no apparent relation to the process of apposition may be seen between these wedges. The condition produced by large scale apposition of cortical tissue differentiating in the capsule has been described elsewhere (Gruenwald and Konikov, '44).

The present material is not sufficient to study changes which may appear in the periphery of the adrenal in old age. This subject will therefore not be discussed.

DISCUSSION

For a proper understanding of the morphology of the adrenal cortex and its changes throughout life, it is important to remember that the organ develops from a mesenchymal primordium, the cells of which are derived from the wall of the coelom including its mesothelium as well as the mesenchyme. For a long period of time the embryonic adrenal cortex remains entirely non-epithelial. Only during the fourth month of intrauterine life the cells of the peripheral zone form epithelial aggregates, as may be demonstrated by the lack of lattice fibers surrounding single cells. The transition between the epithelial and non-epithelial zones remains gradual throughout life with the exception of a temporary demarcation which may appear during childhood.

Collagenous connective tissue fibers are absent from the adrenals of infants except in the capsule and the walls of larger blood vessels. It is, therefore, imperative to impregnate the lattice fibers in order to study the architecture of the adrenal cortex. In the adult, collagenous fibers may reach from the capsule into the cortex for a short distance between the glomerulosa units. Conspicuous bundles or layers of collagenous fibers, extending parallel to the capsule within the cortex, have been interpreted as originating from the capsule and displaced inward by the growth of newly differentiated cortical cell masses.

Embryonic development as reviewed and partly re-examined above produces a condition which is characterized by relatively large glomerulosa units (as compared with the adult) and the absence of forms suggesting apposition of cortical cords newly differentiating in the capsule. During childhood the zona glomerulosa gradually acquires small units which are in most instances direct continuations of single fasciculata cords. At the same time, indications of apposition of cortical tissue from the capsule begin to appear. A relatively common condition in adrenals of older children is tentatively interpreted as a thick layer of newly differentiated cortex laid down over the old zona glomerulosa. Later on, as the appearance of the zona glomerulosa becomes less regular, signs of apposition appear in almost all glands in scattered small areas of the surface.

In a previous study (Gruenwald and Konikov, '44) it was shown that those mammalian adrenals which have a well developed zona glomerulosa with large units usually show less apposition than others with an inconspicuous zona glomerulosa. On the assumption that the zone is a reserve store of cortical cells, this may be interpreted as indicating that organs with a large store need supplies of cells from the outside less frequently than others. The same relationship prevails in human adrenals of varying age; infants' adrenals have a well developed zona glomerulosa and show little apposition of cortical tissue from the capsule. In the adult, on the other hand, the glomerulosa units are small and forms suggestive of apposition are common. The cat is the only species in which the adrenals of young and adult animals have been compared with regard to apposition of cortical tissue from the capsule and the conditions are the opposite of those in man. Young kittens show signs of abundant apposition and a poorly developed zona glomerulosa, whereas mature cats have a larger glomerulosa and, in the author's material, never showed evidence of apposition (Gruenwald and Konikov). It is tempting to relate this difference to the abundance of cortical tissue in the human newborn. Perhaps the hormonal environment of the fetus produces a great excess of this tissue in man and not in the cat; large enough to leave a sufficient margin for the first period of postnatal life even after the degeneration of the x-zone.

An important part of the problem of apposition remains unsolved and that is the time factor. As long as we do not know the speed with which new cortical cells differentiate and become completely incorporated in the main organ, histological findings do not show the actual amount of apposition. This is of great interest, particularly in those adrenals of children which show what is here considered as evidence of very extensive addition of cortical tissue on top of the zona glomerulosa.

Whereas most authors who assume any cell movement in the adrenal cortex at all, conceive of it as proceeding toward the center of the organ, Tonutti has recently advanced a new concept. According to him, differentiation as well as regression of cortical tissue may occur at the boundary of cortex and capsule and simultaneously in the zona reticularis. It is true that the opposite processes of differentiation and degeneration may produce similar forms if one can only examine the condition prevailing at one moment. Yet, in the present findings, the assumption of apposition seems to be better justified, particularly where a zona glomerulosa is seen undisturbed under the layer in question.

Perhaps Tonutti's new views will in the future help explain a condition in children which has no place in our present concept of adrenal architecture and growth. This is the separation of large areas of zona glomerulosa from the underlying cortex by a continuous layer of lattice fibers. It seems that the zona glomerulosa may separate itself temporarily from the fasciculata under conditions not yet understood. Similar observations have been made in cattle embryos and mature cats. Perhaps the zona glomerulosa can, under certain conditions, exist independently, as has been suggested on other grounds by Williams ('45).

It is generally assumed that the endocrine function of the adrenal cortex is related in some manner to the lipoid inclusions in its cells. What conclusions regarding the production or storage of the lipoid-soluble hormone can be drawn from simple fat stains, or various histochemical and physical methods, is an open question. Dosne and Dalton ('41) hold that accumulation of lipoid in cortical cells is in some instances an indication of storage rather than production of hormone, and that very active glands may be depleted of most of their lipoid. A small number of fat stains performed in the course of the present work have again shown the known fact that the zona glomerulosa of children is often very rich in lipoid — more so than the fasciculata. A similar distribution is occasionally found in adults and in various mammals. If, as Sarason ('43) claims, the ordinary sudan stain of frozen sections gives roughly the same results as various more complicated reactions which have been devised to demonstrate certain substances probably related to the specific function, one would have to assume considerable endocrine activity of the zona glomerulosa. This, however, is not in agreement with the concept that the outer portion of the zona fasciculata is the site of hormone production, and the glomerulosa a "presecretory" zone (Bennett, '40) or a reserve store of cortical cells. Perhaps this discrepancy is explained in part by the use of different species by various workers. Also, there is a definite possibility that not all lipoid inclusions in cortical cells have the same value in regard to endocrine function. This is suggested by a difference in the distribution of sudanophil lipoid, ascorbic acid, plasmal, or vitamin A (Fink, '41; Kroczek, '41; Popper, '41).

The pathological significance of cavities within cortical cell cords has recently been emphasized, and previous literature reviewed by Rich ('44). Other cavities which are normally present in the adrenals of human embryos and infants and also in certain adult mammals (da Costa, '28) resemble these pathological cavities to some extent. However, pathological cavities often contain remnants of disintegrating cells which, according to Hett ('25), is never the case in normal embryos. The present specimens show, in accordance with Hett's findings, no cells in embryonic cavities but a fine coagulum. There are, however, adrenals of infants in which it is difficult to decide whether cavities in the zona glomerulosa are of the normal or the pathological type. It has been mentioned that such cavities may correspond to normal ones in their distribution and form but contain cells or debris (fig. 8). It might be of interest in the future to study the two kinds of cavities in their mutual relations.

Accessory cortical cell masses in the vicinity of the adrenal range from groups of cell cords interspersed between the fiber layers of the capsule, to well organized and approximately spherical nodules. The latter resemble the main gland in some respects and differ from it in others. Zones comparable to those of the main gland are present in nodules of sufficiently large size, and degeneration of the x-zone proceeds in the nodules at the same time as in the adrenal itself. Two differences were noted between accessory nodules and the main gland. One concerns the distribution of lipoid. The accessory nodules contain less lipoid, particularly in those cases in which the zona glomerulosa of the main gland is very rich in lipoid. The other difference is apparent only during the first year, and concerns the apposition of cortical cell cords which have newly differentiated in the capsule. This process apparently occurs frequently in the connective tissue surrounding accessory nodules, but not in direct relation to the main gland until later in childhood. It is not known whether the presence of numerous accessory nodules in an infant increases the ability to replace or increase cortical tissue if needed. Even in the adult accessory nodules may behave differently from the main gland, particularly in pathological states. With regard to Addison's disease, Kovacs ('28) and Guttman ('30) hold that cortical nodules may remain undamaged and hypertrophy to a limited extent and maintain life. Rogoff ('45) found in experiments with ligation of adrenal vessels that existing accessory nodules hypertrophy in some, but not in all cases, and supply part of the hormonal requirement.

There are indications that many accessory nodules in the capsule join the main gland when the separating layer of connective tissue is pierced by differentiation of cortical tissue in it. In most instances the nodule loses its independence as soon as it connects with the main gland, as is shown by the disappearance of the zona glomerulosa in the area of contact, and the continuation of the zona fasciculata of the one into that of the other. This is perhaps due to a diversion of the drainage of blood from the nodule toward the main gland as soon as the two come in contact, resulting in a rearrangement of capillaries and cortical cords. In a few cases, however, the nodule seems to retain some degree of individuality after joining the adrenal proper, and forms a distinct group of cords within the cortex. This is not believed to be the only manner in which distinct nodules within the main gland can arise.

SUMMARY

The development of the adrenal cortex from a mesenchymal primordium that was derived from the coelomic lining, as previously described, has been confirmed, and some details have been added. The gland remains entirely non-epithelial until differentiation of the zona glomerulosa begins. This layer is the only one ever to acquire a truly epithelial structure.

At least a large part of the retroperitoneal mesenchyme appears to have the potency to form cortico-adrenal tissue. By differentiation in loco it gives rise not only to the cortex proper in the early embryo, but also to cortical cells in the adrenal capsule and to accessory nodules in later stages.

The zona glomerulosa of the older embryo and the infant consists of relatively wide and tall units in the form of plates or arches, each connecting with many fasciculata cords. These large units are gradually replaced by narrow cords, either by subdivision of the existing units or by apposition of new cords over the old zona glomerulosa. Thus, the well known structure of the mature adrenal evolves; the cords of the zona glomerulosa are mere distal continuations of fasciculata cords and seldom much wider than the latter.

Differentiation of cortical cell cords in the adrenal capsule, and their apposition to the gland itself, does not regularly occur in infants but is frequently seen in older children and adults. This is consistent with comparative results, in mammals, which have shown apposition less frequently in adrenals with a large, well differentiated zona glomerulosa than in those in which that layer is poorly represented.

Accessory cortical nodules in and near the adrenal capsule are a very common, if not a regular occurrence, in infants and young children. They often show indications of active growth by apposition even before this process starts in the main gland. It is believed that many accessory nodules eventually fuse with the main gland.

The cavities which normally appear in the cell groups of the zona glomerulosa of embryos and infants are in some respects similar to pathological cavities in the zona glomerulosa and fasciculata of adrenals of all ages in the course of certain diseases.

The distribution of lipoid in the adrenal cortex is briefly discussed, and it is pointed out that the frequent occurrence of much lipoid in the zona fasciculata is not fully explained by our present concept of cortical morphology and function.

LITERATURE CITED

BACHMANN, R. 1939 Zur Frage der Zona germinativa der Nebennierenrinde. Klin. Wochenschr., vol. 18, pp. 783–784.

BENNER, M. C. 1940 Studies on the involution of the fetal cortex of the adrenal glands. Am. J. Pathol., vol. 16, pp. 787–798.

BENNETT, H. S. 1940 The life history and secretion of the adrenal cortex of the cat. Am. J. Anat., vol. 67, pp. 151–227.

BLUMENTHAL, H. T. 1940 The mitotic count in the adrenal cortex of normal guinea pigs. Endocrinology, vol. 27, pp. 477–480.

——— 1945 Aging process in the endocrine glands of the guinea pig. I. The influence of age, sex and pregnancy on the mitotic activity and the histologic structure of the thyroid, parathyroid and adrenal glands. Arch. Pathol., vol. 40, pp. 264–269.

DA COSTA, A. C. 1922 Structure et histogénèse du cortex surrénal des mammifères. Comptes Rend. Ass. Anat., vol. 17, pp. 79–87.

——— 1928 Les formations vesiculeuses dans les glandes endocrines. Comptes Rend. Ass. Anat., vol. 23, pp. 69–75.

——— 1933 Le cortex surrénal. Bull. Ass. Anat., No. 33.

DIETRICH, A., AND H. SIEGMUND 1926 Die Nebenniere und das chromaffine System. In: Handbuch der speziellen pathologischen Anatomie und Histologie, vol. 8, pp. 951–1089.

DOSNE, C., AND A. J. DALTON 1941 Changes in the lipoid content of the adrenal gland of the rat under conditions of activity and rest. Anat. Rec., vol. 80, pp. 211–217.

FINK, W. 1941 Histochemische Studien über Vitamin C und Plasmalogen an Ovarium und Nebennierenrinde vom Meerschweinchen. Zeitschr. f. mikrosk.-anat. Forsch., vol. 50, pp. 558–589.

GERSCH, I., AND A. GROLLMAN 1939 The nature of the x-zone of the adrenal gland of the mouse. Anat. Rec., vol. 75, pp. 131–153.

GOORMAGHTIGH, N. 1918 Contribution à l'étude du fonctionnement de la capsule surrénale humaine à l'état normal et dans les états infectieux en particulier dans les gangrènes gazeuses. Arch. Méd. Exp., vol. 28, pp. 277–321.

Goormaghtigh, N. 1921 Organogénèse de las capsule surrénale et du plexus coeliaque. Arch. de Biol., vol. 31, pp. 83–172.

Groat, R. A. 1943 Adrenocortical-like tissue in the ovaries of the adrenalectomized ground squirrel (Citellus tridecemlineatus). Endocrinology, vol. 32, pp. 488–492.

——— 1944 Formation and growth of adrenocortical-like tissue in the ovaries of the adrenalectomized ground squirrel. Anat. Rec., vol. 89, pp. 33–41.

Gruenwald, P. 1942a Common traits in development and structure of the organs originating from the coelomic wall. J. Morphol., vol. 70, pp. 353–387.

Gruenwald, P. 1942b The modes of origin of dystopic tissues, with special reference to the problem of hypernephroma. J. Urol., vol. 48, pp. 244–251.

Gruenwald, P., and W. M. Konikov 1944 Cell replacement and its relation to the zona glomerulosa in the adrenal cortex of mammals. Anat. Rec., vol. 89, pp. 1–21.

Guttman, P. H. 1930 Addison's disease. Arch. Pathol., vol. 10, pp. 742–785, 895–935.

Heckel, L. 1942 Untersuchungen über das Vorkommen von Vitamin C in der Nebenniere des Menschen. Zeitschr. f. mikrosk.-anat. Forsch., vol. 52, pp. 393–417.

Hett, J. 1925 Ein Beitrag zur Histogenese der menschlichen Nebenniere. Zeitschr. f. mikrosk.-anat. Forsch., vol. 3, pp. 179–282.

Hill, R. T. 1946 Life sustaining adrenal cortical function of the mouse ovary. Anat. Rec., vol. 94, p. 470.

Hoerr, N. L. 1931 The cells of the suprarenal cortex in the guinea pig. Their reaction to injury and their replacement. Am. J. Anat., vol. 48, pp. 139–197.

Keene, M. F. L., and E. E. Hewer 1927 Observations on the development of the human suprarenal gland. J. Anat., vol. 61, pp. 302–324.

Kohno, S. 1925 Zur vergleichenden Histologie und Embryologie der Nebenniere der Säuger und des Menschen. Zeitschr. f. Anat. u. Entwicklungsgesch., vol. 77, pp. 419–480.

Kolmer, W. 1918 Zur vergleichenden Histologie, Zytologie und Entwicklungsgeschichte der Säugernebenniere. Arch. f. mikrosk. Anat., vol. 91, pp. 1–139.

Kovacs, W. 1928 Zur Nebennierenpathologie. Beitr. z. pathol. Anat., vol. 79, pp. 213–267.

Kroczek, H. 1941 Die Plasmalreaktion in der Nebennierenrinde des Meerschweinchens unter experimentellen Bedingungen. Zeitschr. f. mikrosk.-anat. Forsch., vol. 50, pp. 511–521.

Lewis, R. W., and A. M. Pappenheimer 1916 A study of the involutional changes which occur in the adrenal cortex during infancy. J. Med. Research, vol. 34, pp. 81–94.

Mulon, M. P. 1903 Divisions nucléaires et rôle germinatif de la couche glomérulaire des capsules surrénales du cobaye. Comptes Rend. Soc. Biol., vol. 55, pp. 592–595.

POLITZER, G. 1936 Ueber die Frühentwicklung der Nebennierenrinde beim Menschen. Zeitschr. f. Anat. u. Entwicklungsgesch., vol. 106, pp. 40–48.

POPPER, H. 1941 Histologic distribution of vitamin A in human organs under normal and under pathologic conditions. Arch. Pathol., vol. 31, pp. 766–802.

RICH, A. R. 1944 A peculiar type of adrenal cortical damage associated with acute infections, and its possible relation to circulatory collapse. Bull. Johns Hopkins Hosp., vol. 74, pp. 1–15.

ROGOFF, J. M. 1944 Experimental pathology and physiology of the adrenal cortex. Production of Addison's disease in laboratory animals. Arch. Pathol., vol. 38, pp. 392–409.

SARASON, E. L. 1943 Morphologic changes in the rat's adrenal cortex under various experimental conditions. Arch. Pathol., vol. 35, pp. 373–390.

——— 1943 Adrenal cortex in systemic disease. A morphologic study. Arch. Int. Med., vol. 71, pp. 702–712.

STECKSÉN, A. 1902 Befund von "Adenom" knötchen an Nebennieren und von accessorische Nebennieren bei Erwachsenen. Arb. a. d. Gebiete d. pathol. Anat. a. d. pathol.-anat. Inst. Tübingen, vol. 3, pp. 253–260.

THOMAS, E. 1911 Ueber die Nebenniere des Kindes und ihre Veränderungen bei Infektionskrankheiten. Beitr. z. pathol. Anat., vol. 50, pp. 283–316.

TONUTTI, E. 1941 Hormonal gesteuerte Transformationsfelder in der Nebennierenrinde? Zeitschr. f. mikrosk.-anat. Forsch., vol. 50, pp. 495–501.

——— 1942a Zur Histophysiologie der Nebennierenrinde: Bau und Histochemie bei der Atrophie des Organs nach Hypophysektomie. Zeitschr. f. mikrosk.-anat. Forsch., vol. 51, pp. 346–392.

——— 1942b Die Umbauvorgänge in den Transformationsfeldern der Nebennierenrinde als Grundlage der Beurteilung der Nebennierenarbeit. Zeitschr. f. mikrosk.-anat. Forsch., vol. 52, pp. 32–86.

UOTILA, U. U. 1940 The early embryological development of the fetal and permanent adrenal cortex in man. Anat. Rec., vol. 76, pp. 183–203.

WARING, H. 1935 The development of the adrenal gland of the mouse. Quart. J. Microsc. Sci., vol. 78, pp. 329–366.

WHITEHEAD, R. 1943 The growth of the adrenal cortex in the guinea pig. J. Pathol. and Bact., vol. 55, p. 392.

WILLIAMS, R. G. 1945 The characteristics and behavior of living cells in autogenous grafts of adrenal cortex in rabbits. Am. J. Anat., vol. 77, pp. 53–79.

WOTTON, R. M., AND R. L. ZWEMER 1943 A study of the cytogenesis of cortico-adrenal cells in the cat. Anat. Rec., vol. 86, pp. 409–416.

ZWEMER, R. L., R. M. WOTTON AND M. G. NORKUS 1938 A study of cortico-adrenal cells. Anat. Rec., vol. 72, pp. 249–263.

PLATE 1

EXPLANATION OF FIGURES

1–3 Adrenals of human embryos of 67, 145 and 170 mm crown-heel length, respectively. 1 and 2, Gomori's silver impregnation; 3, hematoxylin-eosin. In contrast to the early stage (fig. 1), figure 2 shows a peripheral layer of the cortex in which individual cells are not surrounded by lattice fibers. Figure 3 shows numerous cavities and cells of small size in this peripheral layer.

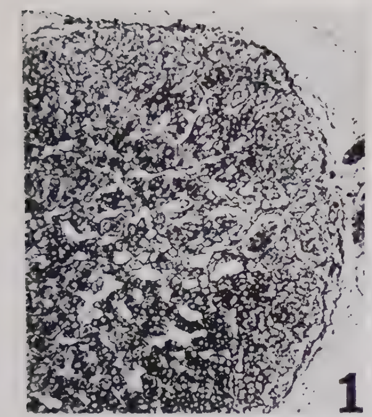
4 Adrenal of a 2-day-old female infant with a zona glomerulosa consisting of large units which appear on section as arches. Gomori's silver impregnation.

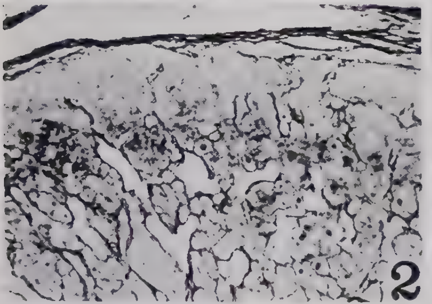
5 Adrenal of a 15-day-old male infant. Gomori's silver impregnation. The large units of the zona glomerulosa are being subdivided into narrow cords by new lattice fiber septa.

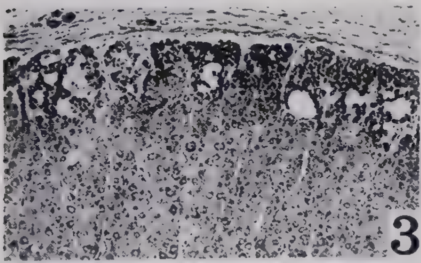
6–7 Adrenal of a 2½-month-old infant. 6, Gomori's silver impregnation; 7, azan stain. The zona glomerulosa contains very wide arches. Figure 7 shows the transition of small cells in the zona glomerulosa (right half of figure) into large cells (left half).

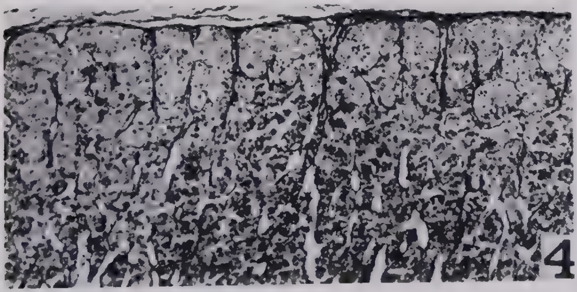
8 Adrenal of a 6-month-old female infant. Azan stain. The zona glomerulosa contains many cavities.

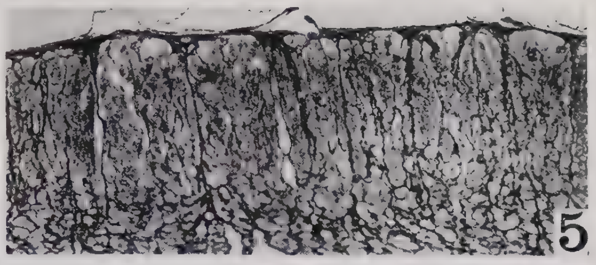
9 Adrenal of a boy of 1 year and 7 months. Gomori's silver impregnation. The zona glomerulosa consists of thick cell cords, and is almost completely separated from the deeper layers by lattice fibers.

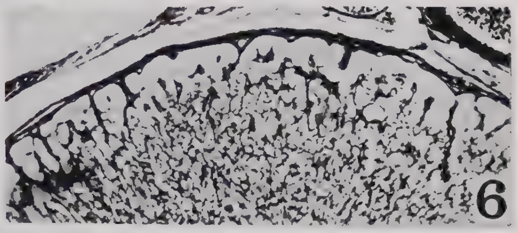

1

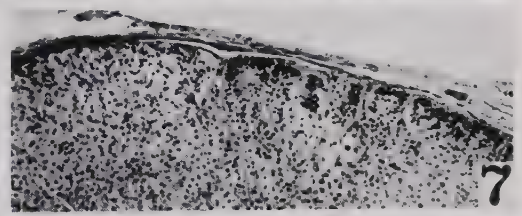

2

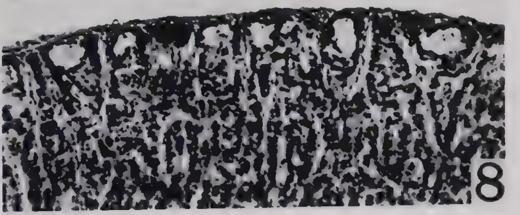

3


4


5


6


7


8

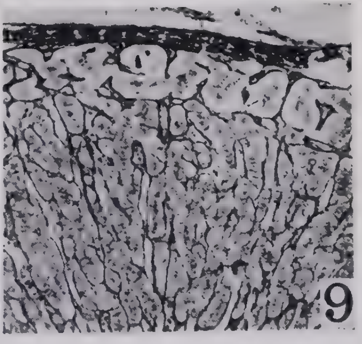

9

PLATE 2

EXPLANATION OF FIGURES

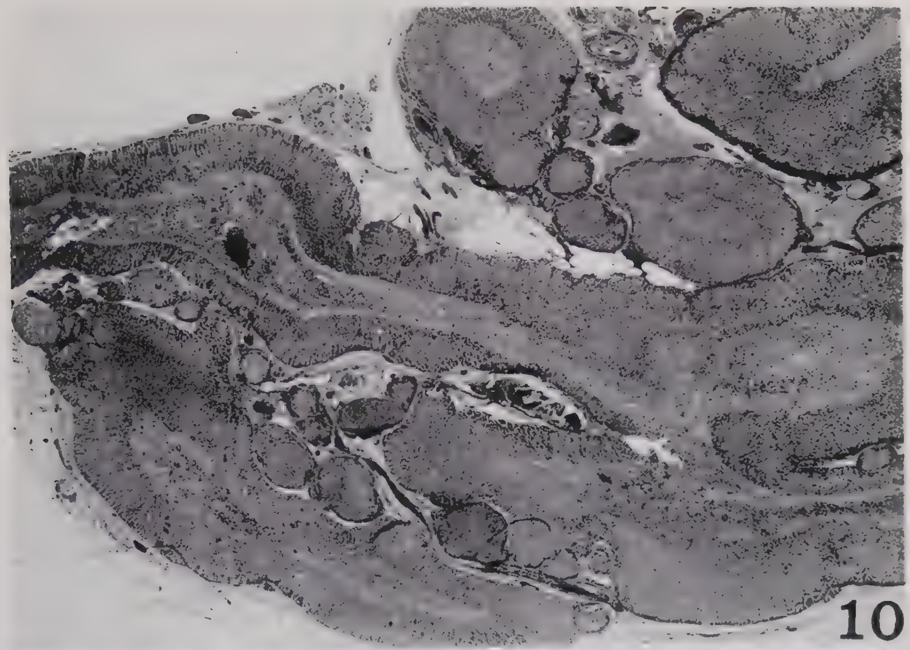
Accessory cortical nodules

10 Adrenal of a 3½-month-old male infant, showing numerous accessory nodules. Azan stain.

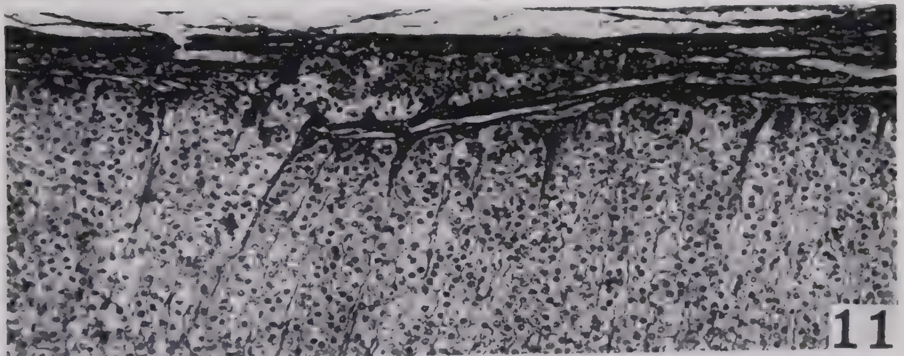
11 Adrenal of a 2½-month-old infant (same as in figs. 6 and 7) with a mass of accessory cortical tissue in the capsule, connecting at one point with the main organ. Van Gieson stain.

12 Accessory nodule in the adrenal capsule of a 4-month-old male infant. Hematoxylin-eosin stain. The capsule of the nodule contains cortical cell cords in tangential arrangement between its fiber layers (arrows).

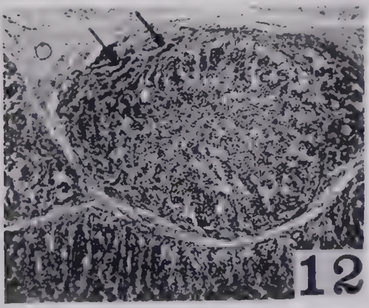
13 Nodule in the epididymis of a 2-month-old infant. Hematoxylin-eosin stain. There are similar cell cords in the capsule as in figure 12. The right side of the figure shows the region of the degenerating x-zone.



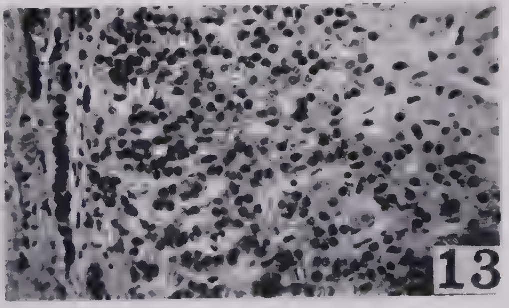
10



11



12



13

PLATE 3

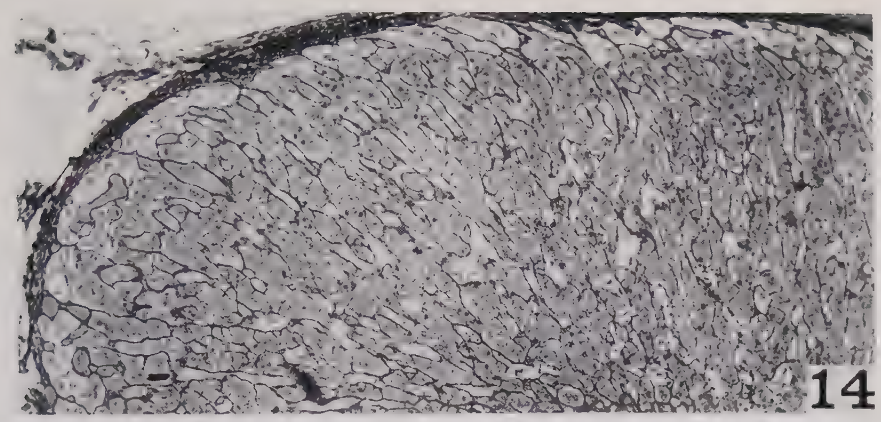
EXPLANATION OF FIGURES

14 Adrenal of a boy of 2 years and 8 months. Gomori's silver impregnation. Many glomerulosa cords have tapering distal ends which bend and approach the direction of the capsule.

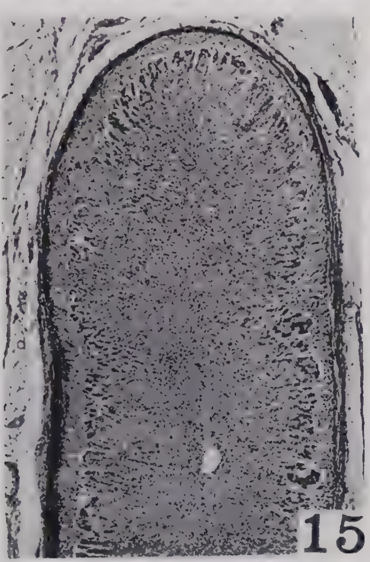
15–16 Adrenal of a 6-year-old girl. 15, Van Gieson stain; 16, Gomori's silver impregnation, higher magnification. There is a layer of thin cortical cell cords between the capsule and large arches usually seen in the zona glomerulosa.

17 Adrenal of a 4-year-old boy. Azan stain. The peculiar arrangement of a group of cell cords with the cortex is explained in the text.

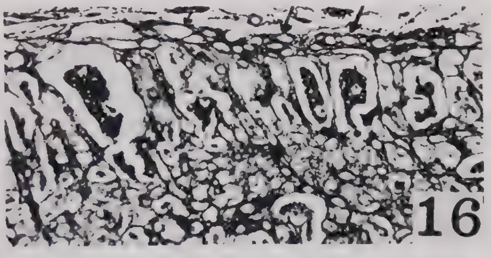
18 Adrenal of a 59-year-old man. Gomori's silver impregnation. For an explanation of the course of the cell cords in the cortex, see the text.



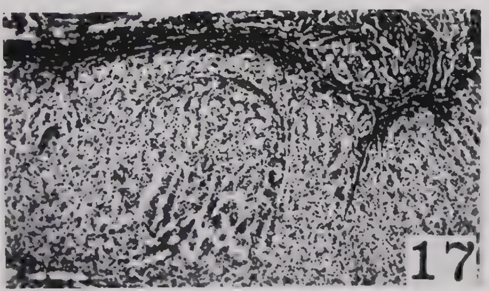
14



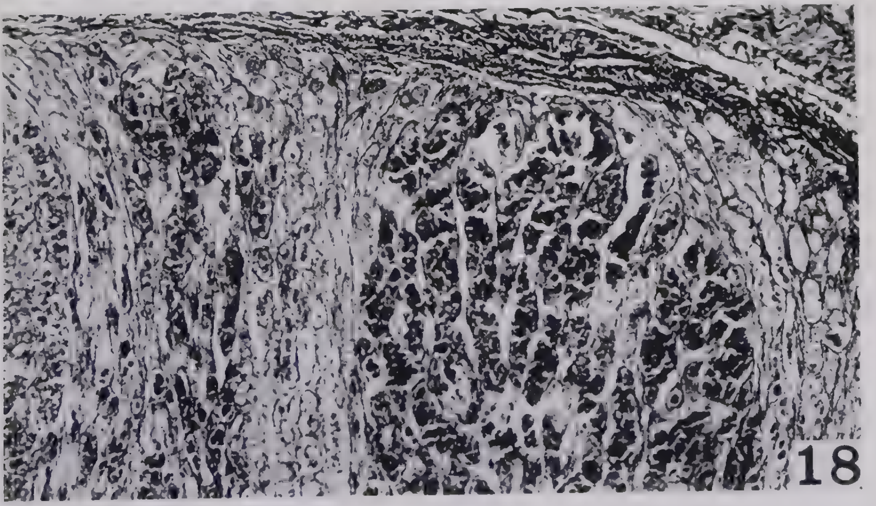
15



16



17



18

A SIMIAN, DEEPLY CLEFT, BILOBED GALL BLADDER WITH A "PHRYGIAN CAP"

HADLEY KIRKMAN
Department of Anatomy, Stanford University School of Medicine, California

THREE TEXT FIGURES AND TWO PLATES (THIRTY-FOUR FIGURES)

Among the anomalies of the human biliary tract the most frequent is a gall bladder with a fundus folded in such a manner as to resemble a "phrygian cap" (Bartel, '16, '18). This anomaly was discussed thoroughly by Boyden ('35) who found 18% of 165 individuals to possess it. The rarest of these anomalies consists of a gall bladder partially subdivided by a longitudinal septum, but drained by a single cystic duct (vesica fellea divisa). The comparative anatomy and embryology of this, and of other types of gall bladder anomalies, was presented in detail by Boyden ('26), who reported slightly under 0.016% bilobed gall bladders (three cases) in a series of 19,000 observations collected by him. Of these three reports only one included an adequate description. It is unfortunate that many writers, e.g. Stolkind ('40) in his recent review, have included as bilobed gall bladders specimens in which a transverse septum, or constriction, has resulted in the formation of two successive compartments. These reports are not considered in this paper. Certain other cases referred to as bilobed, on the basis of cystographic examination alone, would be better classified as instances of the "phrygian cap" anomaly, and so are omitted from the summary of reported instances of bilobed gall bladders presented in table 1.

Variation in the form of monkey gall bladders was noted as early as 1862 (Crisp), but his observations received scant attention from subsequent investigators. Little is known concerning the incidence of gall bladder anomalies in subhuman primates (table 2). Friedlowsky, in 1870, gave a brief, unillustrated, description of what may have been a deeply cleft, bilobed gall bladder in a monkey (Macacus cynomolgus). Since the septum was said to end at the beginning of the cystic duct, there is some possibility of the specimen having been a double bladder. This possibility is suggested further by Friedlowsky's comment on having seen a similar human specimen stored in the museum of pathological anatomy in Vienna. Halpert ('27a) mentioned incidentally that one of twenty-five monkey (Macaca rhesus) gall bladders examined was bilobed. In 1929 he published a photograph of a trilobed specimen (reproduced also by Weiss, '35) in the same species. Although there was no accompanying description and while I was unable to discern either cystic duct or ductus choledochus in the illustration, the specimen, in all likelihood, was a genuine example of a trilobed, cleft, gall bladder (vesica fellea divisa). Tittler ('45), in 158 rhesus monkeys dissected, found one gall bladder which was probably bilobed, although there is some doubt about its classification. These scant data might suggest that bilobed gall bladders may be much more common among monkeys than among men. On the other hand neither Rex (1888), nor Ruge ('02a, '02b, '06a, '06b, '07, '08) in their extensive studies of primate livers, mentioned having encountered a bilobed gall bladder. The anomaly has been reported also in species other than primate, e.g. aardvark? (Jaeger?, from Rau, '28); buffalo calf (Mella, from Boyden, '26); calf (Ruysch, 1721; Boyden, '26); cat (Boyden, '26); giraffe (Owen, 1868); lion (Halpert, '27a); pig (Boyden, '26); sheep (Boyden, '26).

In view of the scarcity of available descriptions of monkey gall bladders (Ruch, '41 lists only three references in his Bibliographia primatologica), and because of this reported rarity of bilobed gall bladders among primates, it is considered worthwhile to present in some detail the specimen described below.

TABLE 1

Reported instances of human, bilobed, gall bladders.

AUTHOR	YEAR	SEX	AGE	NORMAL OR PATHOLOGICAL	BASIS OF DESCRIPTION	REMARKS
Cruveilhier	1865				Gross specimen	Incidental illustration; no comment in legend or text!
Friedlowsky	1870				Gross specimen	Doubtful; no illustration; no description.
Deaver and Ashhurst	1914	♀	55	Pathological	Gross specimen	Doubtful; omitted by Deaver and Ashurst in their second (1921) edition.
Rycroft	1923	♀		Pathological	Gross and microscopic specimens	Doubtful, due to marked pathology; no description of cystic duct. Accepted as bilobed by Gross, '36 and Stolkind, '40.
Boyden	1926			Normal	Cholecystograph confirmed at operation.	Ullmann's specimen; genuine bilobed gall bladder.
Levyn and Aaron	1926				Cholecystograph	Doubtful; unrecognized by authors; see Boyden, '26, p. 205 for discussion.
Meyer	1928			Normal	Gross specimen	Lobes communicated with each other, but only one lobe communicated with duct. Possibly a true bilobed gall bladder, but uncertain. Accepted as bilobed by Stolkind, '40.
Phillips, Isaac and McDonald	1931	♂		Pathological	Gross specimen	Possibly a bilobed specimen; one lobe buried in a mass of adhesions. Accepted as bilobed by Gross, '36 and Stolkind, '40.
Weiss	1935				Cholecystograph	2 cases, inadequately described. Possibly bilobed.
Belot and Nahan	1937				Cholecystograph	Doubtful; photographs suggest ''phrygian cap,'' rather than bilobed gall bladder.
Hilt	1937	♀			Cholecystograph confirmed at operation.	Description inadequate. Possibly bilobed. Accepted as bilobed by Stolkind, '40.
Stolkind	1940	♂		Normal	Gross specimen	Septum perforated by orifice connecting lobes; no description of cystic duct. Possibly bilobed, but doubtful.

TABLE 2

Reported instances of possible anomalies in the extrahepatic biliary tracts of subhuman primates.

AUTHOR	YEAR	SPECIES	SEX	AGE	NORMAL OR PATHOLOGICAL	BASIS OF DESCRIPTION	TYPE OF ANOMALY	REMARKS
Drelincourt	?	Ape	♀			Gross specimen	No ampulla of Vater	Ampulla in ♂ ape (quoted by Blasius, 1681 according to Tyson, 1699).
Perrault	1688	Monkey				Gross specimen	No ampulla of Vater	Common opening in apes (from Tyson, 1699).
Perrault	1688	Monkey				Gross specimen	No common hepatic duct	(from Tyson, 1699.)
Tyson	1699	Monkey				Gross specimen	No ampulla of Vater	Common opening in ape and "orang-outang" (according to Gratiolet and Alix, 1866, Tyson's orang-outang was a chimpanzee).
Daubenton	1766	Marmoset (Hapale jacchus)				Gross specimen	Absence of gall bladder	Doubted by Meckel, 1838 (quoted by Flower, 1872 from Buffon, 1766).
Cuvier	1799	Sapajous and baboon (Papio inuus)				Gross specimen	No ampulla of Vater	Questioned by Meckel (1838).
Meckel	1838	Spider monkey				Gross specimen	No ampulla of Vater	
Sandifort	1840	Orang-utan				Gross specimen	No ampulla of Vater	Agrees with Chapman, 1880 and Straus, '36, but not with Gratiolet and Alix, 1866, Anthony, 1898, and Sonntag, '24.
Friedlowsky	1870	Monkey (Mac. cynomolgus)		Young		Gross specimen	Bilobed gall bladder	Possibly double gall bladder.
		(Cercopithecus sabaeus)					Ductular type of double gall bladder	Illustrated and described.

TABLE 2 — *(continued)*

AUTHOR	YEAR	SPECIES	SEX	AGE	NORMAL OR PATHOLOGICAL	BASIS OF DESCRIPTION	TYPE OF ANOMALY	REMARKS
Flower	1872	Gibbon			Normal	Gross specimen	No common hepatic duct	p. 394, fig. 12.
		Lemur, (Microcebus)			Normal	Gross specimens	Accessory hepatic ducts entering cystic duct. Gall bladder reversed in position.	p. 562, fig. 20.
Robinson	1907	Baboon				Gross specimen	No common hepatic duct	Long cmomon duct in monkey.
Sonntag	1923	Chimpanzee			Normal	Gross specimen	Embedded gall bladder.	Not characteristic, but observed in occasional specimens.
Halpert	1927a and 1929	Monkey (Mac. rhesus)			Normal	Gross specimens	Bilobed gall bladder. Embedded gall bladder.	
Rau	1928	Monkey (Loris lydekkerianus)				Gross specimen	Triplicate gall bladder.	Ductular type.
Halpert	1929	Monkey (Mac. rhesus)			Normal	Gross specimen	Trilobed gall bladder.	Photograph without description. Illustrated also in Weiss, '35, p. 38.
Straus	1936	Orang-utan	♀	Infant	Normal	Gross specimen	No ampulla of Vater	
	1937	Chimpanzee	♀	74 days	Normal	Gross specimen	No ampulla of Vater	
Tittler	1945	Monkey (Mac. rhesus)				Gross specimens	Diverticular gall bladder with "phrygian cap." Ductular gall bladder.	Possibly bilobed gall bladder.

MATERIALS AND METHODS

The biliary tract described in this paper was taken from a female rhesus monkey (Macaca mulatta; no. 267). On the basis of sitting height (440 mm), head length (120 mm), foot length (140 mm), leg length (150 mm), tail length (225 mm), and dentition (all permanent teeth, except upper and lower third molars erupted) she was estimated to be about 7 years of age (Schultz, '33). She was sacrificed after receiving heavy estrogen injections. Under anaesthesia the blood was washed out quickly with saline solution, followed by Bouin's fluid. The perfused animal was then stored in 10% formalin until used. The biliary tract was dehydrated in ethyl alcohol and embedded in amyl acetate nitrocellulose. The blocks were sectioned at 12 μ and 14 μ; six sections at 12 μ and one hundred at 14 μ, etc. Representative sections from successive levels were stained with hematoxylin and eosin, Mallory azan, Verhoeff's elastic fiber stain, and Foot's silver ammonium carbonate for reticular fibers. To obtain satisfactory results with Foot's technique it was found necessary to mount the sections on slides and remove all of the celloidin, since the amyl acetate, unlike ether-alcohol, interfered with the silver impregnation.

For the most part measurements of the biliary tract were made upon the fixed material, in 70% alcohol. In some instances, e.g. diameters of ductus cysticus and choledochus and of ductuli hepaticae, measurements were made from sections projected on paper and traced at eight diameters magnification. Corrections for shrinkage were made from the data supplied by Stowell ('41) and by the method described by Kirkman and Stowell ('42).

Various portions of the biliary tracts of six other rhesus monkeys were studied for comparison with the one described here (table 3).

OBSERVATIONS

Viewed macroscopically, in situ, after perfusion, the gall bladder showed a narrow yellow band (cystic artery) bordered by green, extending longitudinally along the serosal surface from fundus to about the middle of the infundibulum (fig. 1, S). This line marked the attachment of the underlying septum. A portion of the serosal surface of the folded fundus also, was visible under the inferior border of the right central lobe of the liver (PC in figs. 1, 5 and 6). Otherwise the gall bladder appeared normal. The fossa vesicae felleae involved the greater portion of the inferior surface of the right central lobe. On the superior surface of the right central lobe, at the inferior border, an oval area of the "cap," measuring 15 mm

TABLE 3

Portions of the extrahepatic biliary systems of rhesus monkeys used as controls for monkey number 267.

ACC. NO.	SEX	AGE	PREVIOUS HISTORY	HEPATIC DUCTS	CYSTIC DUCT	COLLUM	CORPUS	DUCTUS CHOLEDOCHUS	AMPULLA OF VATER
158	♂	Young	Used for "Polio" innoculation				+		
170	♀		Suffered from tuberculosis	+	+	+	+		
178	♀		Suffered from tuberculosis				+		
201	♀	18 days old	Cause of death undetermined	+	+	+	+		+
205	♂							+	+
277	♀			+	+	+	+	+	+
MY105	♀		Had been used for antuitrin S injections	+	+	+	+		

× 9 mm, was covered only by serosa (LS in figs. 5 and 6) and constituted the only indication of a pars libera (Nuboer, '31). Straus ('36) too, observed a ventrally exposed gall bladder in one specimen of Macaca mulatta, although among primates he considered this condition to be characteristic of lemurs alone. Length and breadth of the gall bladder may be determined by measurements on figure 1. The greatest depth was 10 mm and the thickness of the septum 1 mm. The average width of the left compartment was only slightly less than that of the right one; nevertheless, its capacity was considerably less, since the large "phrygian cap" was formed exclusively by the fundus of the right compartment (figs. 6 and 7). The fundus of the larger compartment communicated with the corpus through a constricted isthmus having diameters of 6 and 7 mm (fig. 7). The folded fundus was clearly of the serosal type (Boyden, '35), the peritoneum following the bend closely, much as the pia mater follows the convolutions of the cerebrum.

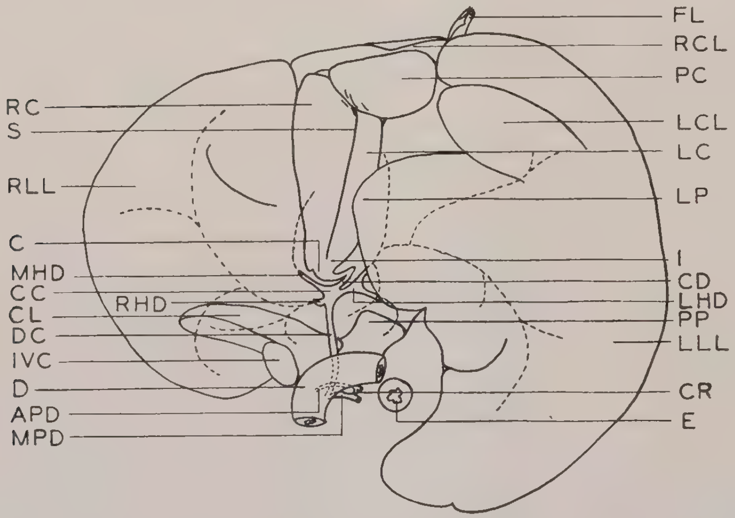


Fig. 1 Inferior surface of liver and extra-hepatic biliary tract including bilobed gall bladder; broken lines indicate intra-hepatic course of bile ducts. For explanation of abbreviations see page 442. Magnification 0.8 ×.

As seems to be common with material fixed by vascular perfusion (Halpert, '27a, fig. 10; Pfuhl, '32, fig. 9; Maximow and Bloom, '42, fig. 377) the gall bladder appeared in the distended state. In the fundus and corpus the mucosal folds were low, but easily apparent to the unaided eye (figs. 7, 8 and 9). Their inner surface pattern agreed essentially with that described by Halpert ('27a) for the normal human gall bladder. In the infundibulum, on the other hand, the mucosa appeared entirely smooth, except for three or four very poorly developed spiral folds (figs. 2, 10, 11, 12 and 13).[1] The transition between these two mucosal surfaces was quite abrupt around the entire circumference of each compartment. Only the distal half of the infundibulum, i.e. the half toward the fundus, was subdivided, the two compartments communicating directly with the undivided proximal half (figs. 1 and 2). The infundibulum terminated abruptly at the collum which continued to the left, at right angles to the axis of the corpus and infundibulm (figs. 1 and 13). A lymph node lay against the inferior surface of the infundibulum at its junction with the collum (fig. 14). The external surface of the collum was somewhat tortuous due to the internal spiral folds. The union of collum and cystic duct was marked by an abrupt U-shaped turn. The short cystic duct was joined by the middle hepatic duct (Ruge, '02a) draining the right portion of the right central lobe (figs. 1 and 15) to form a very short common chamber (fig. 16). Almost immediately the left hepatic duct, draining the left portion of the right central lobe, the left central lobe, the left lateral lobe and the papillary process of the caudate lobe, emptied into this chamber from the left (figs. 1, 16 and 17). On the right, the right hepatic duct, draining the right lateral lobe and the caudate lobe, promptly terminated in the common cavity marking the beginning of the ductus choledochus (figs. 1 and 18). There was, therefore, no common hepatic duct. Since the absence of a common hepatic duct was not mentioned by Lineback ('33), in his description of the biliary tract of the rhesus monkey, these ducts were dissected out in another

---

[1] The collum (neck) of the gall bladder is usually defined (Poirier and Charpy, '01; Aschoff, '24; Boyden, '35) as the portion between the first spiral fold and the cystic duct, demarcated at each end by an acute bend. According to this definition the portion which I describe as infundibulum might have been a dilated distal portion of the collum (cf. Halpert, '29), providing the spiral folds are considered abortive valves of Heister; however, its size, shape, demarcations from adjacent regions, absence of glands and atrophic appearance of spiral folds (fig. 2) appeared to justify identifying it as infundibulum rather than as collum.

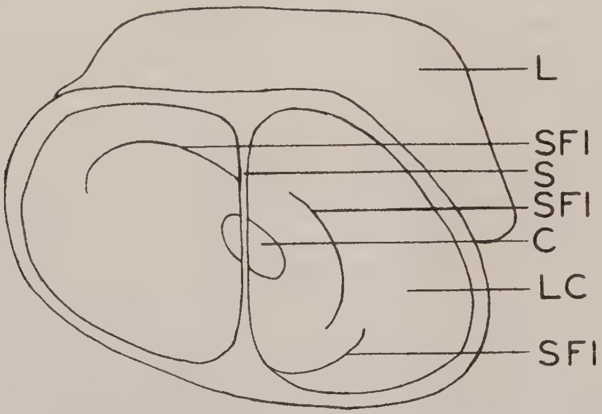


Fig. 2 View of divided infundibulum and undivided collum at level indicated on figure 4. The only mucosal folds present were the slightly elevated spiral ones (see also fig. 12). For explanation of abbreviations see page 442. Magnification 4.2 ×.

rhesus macacque (fig. 3). In this specimen (no. 277, a very large female)the cystic duct joined the three hepatic ducts to form a short, common chamber terminating immediately in the ductus choledochus, a condition differing but slightly from the one described above. Among the earliest descriptions of comparable arrangements of these ducts in the monkey, was that of Perrault (1676), denied by Robinson ('07). Robinson however, found no common hepatic duct in the baboon; Flower, 1872, found none in the gibbon. The numerous variations in hepatic ducts described by Ruge ('02a) did not include this condition.

From the porta hepatis to the duodenum the ductus choledochus ran first through the ligamentum hepatoduodenale (fig. 19) to the caput pancreatis, which formed a ring about it (fig. 21) throughout the remainder of its extramural course, as in man. As it reached the tunica muscularis duodeni it was joined by the ductus pancreaticus major (figs. 22 and 23). The two continued side by side obliquely through the muscularis on the medial side of the duodenum. As they entered the tela submucosa, a plica longitudinalis duodeni first became apparent (figs. 28 and 29). The ducts continued through the submucosa a distance of slightly more than 4 mm before penetrating the muscularis mucosae. They had a common lumen during the last 3 mm of this distance (figs. 32 and 33). Nevertheless the mucosa of the common duct retained, to some extent, throughout much of the remainder of its course, the two slightly different types of mucosal folds characteristic of the terminal portions of the parent tributaries (figs. 29, 30, 31,

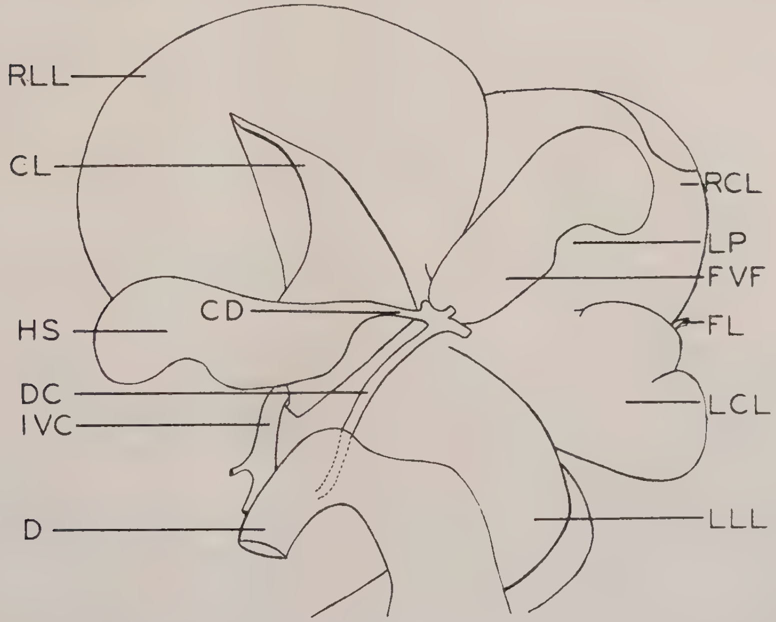


Fig. 3 Inferior surface of liver and fossa vesicae felleae from control monkey (no. 277). The gall bladder has been detached from the fossa and reflected backward. The three hepatic ducts join with the cystic duct to form a short common chamber. For explanation of abbreviations see page 442. Magnification 1 ×.

and 34). The differences were similar to those described for the cat (Boyden, '25). As in the dog, (Hendrickson, 1898; Boyden and Schwegler, '36) separate openings for the two ducts have been reported for certain primates, e.g. female ape (Drelincourt, 1681; denied by Perrault, 1688; and by Tyson, 1699); monkey (Perrault, 1688; Tyson, 1699); sapajous and baboon (Cuvier, 1799; questioned by Meckel, 1838); spider monkey (Meckel, 1838); and orang-utan (Sandifort, 1840; Chapman, 1880; and Straus, '36; contrary to Gratiolet and Alix, 1866; Anthony, 1898 and Sonntag, '24). Numerous reports on the chimpanzee have agreed that in this species the two ducts have a common aperture.

A sphincter started about the ductus choledochus 3½ mm before the duct made contact with the muscularis duodeni (near level represented by fig. 21). It attained its greatest development just outside the circular stratum of the duodenal muscular tunic (figs. 22, 23, 24, 26, and 28). After penetrating the muscularis duodeni it thinned out markedly (figs. 28, 29 and 30) before the beginning of the ampulla. A sphincter pancreaticus existed only around the terminal 2 mm of the pancreatic duct (figs. 23, 26, 27, and 31). As the two ducts united in the ampulla the thinned out sphincters were replaced, as in man (Boyden, '37; Kreilkamp and Boyden, '40) by the very poorly developed sphincter ampullae (figs. 30, 31, 32, 33, and 34) which disappeared before the ampulla reached the muscularis mucosae of the duodenum. Longitudinal muscle fibers associated with the three ducts were inconspicuous. Due to the difficulty of interpreting the detailed structure of the very complicated musculature of the so-called sphincter of Oddi from serial sections no attempt was made to compare the sphincter of Oddi of this monkey with those so carefully studied in human macerated preparations and in serial sections by Boyden ('36, '37) and his associates (Schwegler and Boyden, '37a, '37b, '37c; Kreilkamp and Boyden, '40). However, certain differences between human and monkey material are conspicuous enough to warrant mention. Whereas in adult man the ampullary segment is very short, due to involution

(Boyden, '37), in the monkey it is quite prominent. In human material reinforcing fibers, from the duodenal musculature to the sphincter of Oddi, are numerous but delicate. Certain of them, as recognized also in the chimpanzee fetus by Kreilkamp and Boyden ('40), are especially well developed in the monkey (figs. 24, 25, 26, 27). In man the sphincter choledochus is best developed after the duct has entered the duodenal submucosa; in the monkey its maximum development occurs during penetration of the duodenal muscularis (figs. 22, 23, 28, 29).

Similarity of the extrahepatic biliary tracts of man and monkey, in all essential histological features, is to be expected, since the monkey (Macaca rhesus) is the only species known to have an initial emptying rate for the gall bladder comparable to that of man (Boyden, '32).

About 4½ mm to the right and slightly superior to the intestinal aperture of the common duct (fig. 35) was a small conical papilla pierced by the ductus pancreaticus accessorius (figs. 36 and 37). By tracing the accessory duct through the head of the pancreas it was found to give rise to a tributary communicating with the major duct as mentioned by Lineback ('33) (fig. 1). According to Patzelt ('36), Stracker ('09) observed several openings of the accessory duct into the duodenum of the monkey.

Average diameters of various levels of the biliary tract are listed in table 4. The figures given by Mann, Brimhall and Foster ('20) for cystic duct and ductus choledochus agree quite well, their average for the former being 0.1 mm less and for the latter 0.2 mm greater than given in table 4. The table includes, also, the thickness of the epithelial layers in different levels of the tract.

The histology of the extrahepatic biliary system of this monkey was similar to that of the control monkeys, except for the bilobed condition of the gall bladder, the "phrygian cap," the small area of ventrally exposed gall bladder and the abrupt transition from a folded to a smooth mucosa in the infundibulum. No true Luschka ducts nor Rokitansky-Aschoff sinuses

(Halpert, '27b) were observed around the gall bladder, but diverticula comparable to the sinuses were seen frequently about the ductus choledochus (figs. 24 and 25). No adequate description of the histology of the extrahepatic biliary tract appears to be available for any primate other than man; however, the monkey material reported in this paper disclosed no essential differences from human material (Pfuhl, '32) other than those specifically mentioned.

TABLE 4

Average diameters and epithelial cell heights, in millimeters, of various levels of the extrahepatic tract, and of related structures, of Macaca mulatta no. 267. All measurements corrected for shrinkage.

ORGAN	AVERAGE DIAMETER	AVERAGE THICKNESS OF EPITHELIUM
Corpus	25.00	0.047
Infundibulum	12.00	0.038
Collum	7.50	0.038
Cystic duct	2.57	0.054
Middle hepatic duct	1.00	0.035
Left hepatic duct	1.71	0.043
Right hepatic duct	1.56	0.038
Common chamber	3.28	0.043
Ductus choledochus	2.85	0.044
Major pancreatic duct		
(about 2 mm from ductus choledochus)	1.71	0.040
(in duodenal submucosa)	2.28	0.040
Ampulla of Vater	2.85	0.040
Accessory pancreatic duct	0.71	0.034
Duodenal villi		0.084

The comparative anatomy and embryology of the cleft gall bladder was presented in detail by Boyden ('26). It need only be mentioned here that the anomaly is believed to originate as an initial subdivision of the primary cystic diverticulum of the embryo. While rare in man it is very common among cats (90% of 315 cases of accessory gall bladders found in 2568 individuals).[1] The prevailing type of accessory gall bladder in man is the vesica fellea duplex, or double gall bladder with two cystic ducts; this type is rare among cats (7% of 315 accessory gall bladders). Other types of biliary anomalies were classified and reviewed briefly by Boyden ('44).

---

[1] A personal communication from Professor Boyden informs me that his statistics, based upon Boston cats, do not apply to the Minneapolis region. Of the several hundred Minnesota cats examined by him, he can hardly recall one that had a bifid or a double gall bladder.

According to Boyden ('26), in the majority of bilobed gall bladders the left lobe is better developed than the right. In the case described here the reverse was true.

SUMMARY

The macroscopic anatomy of an accessory gall bladder in an adult, female, rhesus monkey is described. The anomaly is classified as a deeply cleft, bilobed gall bladder (vesica fellea divisa). The two lobes were adherent. The right lobe was the larger of the two and its fundus was folded over that of the left lobe to form a second anomaly commonly referred to as a "phrygian," or "liberty cap." The folded fundus was of the serosal type. The rest of the extrahepatic biliary system included no common hepatic duct; the cystic duct and the three hepatic ducts terminated separately in a very short common chamber which continued as the ductus choledochus. The ampullary segment of the ductus choledochus and certain reinforcing fibers from the duodenal musculature to the sphincter of Oddi were more prominent than in human material, otherwise the system was macroscopically and microscopically essentially similar to that of man.

LITERATURE CITED

ANTHONY, R. 1898 Mémoire sur les organes viscéreaux d'un jeune orang-outan femelle. Bull. Soc. Anthrop., Paris, vol. 9, ser. 4, pp. 246–270.

ASCHOFF, L. 1924 Lectures on pathology. VIII. Orthology and pathology of the extra-hepatic bile passages. Hoeber, N. Y.

BARTEL, J. 1916 Cholelithiasis und Körperkonstitution. Frankfurt. Ztschr. f. Path., vol. 19, pp. 206–237.

——— 1918 Ueber eine Formanomalie der Gallenblase und ihre biologischen Beziehungen. Wien. klin. Wchnschr., vol. 31, pp. 605–611.

BELOT, J., AND L. NAHAN 1937 La radiologie du practicien. Presse Méd., vol. 45, pp. 639–640.

BLASIUS, G. 1681 Anatome animalium. Amstelodami (from Tyson, 1699).

BOYDEN, E. A. 1925 The problem of the pancreatic bladder — a critical survey of six new cases, based on new histological and embryological observations. Am. J. Anat., vol. 36, pp. 151–183.

BOYDEN, E. A. 1926 The accessory gall-bladder. An embryological and comparative study of aberrant biliary vesicles occurring in man and the domestic mammals. Am. J. Anat., vol. 38, pp. 177–231.

——— 1932 Emptying of the gall bladder in monkeys. Proc. Soc. Exp. Biol. Med., vol. 29, pp. 1104–1105.

——— 1935 The "phrygian cap" in cholecystography. A congenital anomaly of the gall bladder. Am. J. Roentgenol. and Rad. Therapy, vol. 33, pp. 589–602.

——— 1936 The pars intestinalis of the common bile duct as viewed by the older anatomists (Vesalius, Glisson, Bianchi, Vâter, Haller, Santorini, etc.). Anat. Rec., vol. 66, pp. 217–232.

——— 1937 The sphincter of Oddi in man and certain representative mammals. Surg., vol. 1, pp. 25–37.

——— 1944 Congenital variations of the extrahepatic biliary tract. A review. Minn. Med., vol. 27, pp. 932–933.

BOYDEN, E. A., AND R. A. SCHWEGLER, JR. 1936 The sphincter of Oddi in the dog (abst.). Am. J. Physiol., vol. 116, p. 14.

BUFFON, G. L. L. DE 1766 Histoire naturelle, vol. XV, 102, Paris.

CHAPMAN, H. C. 1880 On the structure of the orang-outang. Proc. Acad. Nat. Sci., Philadelphia, pp. 160–175.

CRISP, E. 1862 On the situation, form, and capacity of the gall bladder in the vertebrata; on its absence in certain animals; and on the color of the bile. Proc. Zool. Soc., Lond., pp. 132–139.

CRUVEILHIER, E. 1865 Traité d'anatomie descriptive, vol. 2.

CUVIER, G. 1799 Lecons d'anatomie comparée. 2nd ed., vol. IV, 2nd part, Paris (from Meckel, 1838).

DAUBENTON (quoted by Flower 1872, from Buffon 1766).

DEAVER, J. B., AND A. P. C. ASHHURST 1914 Surgery of the upper abdomen, vol. 2, p. 42. Blakiston, Philadelphia.

DRELINCOURT (from Tyson, 1699).

FLOWER, W. H. 1872 Lectures on the comparative anatomy of the organs and digestion of the mammalia. The Medical Times and Gazette, vol. 62.

FRIEDLOWSKY, A. 1870 Ueber gelappte Gallenblase bei einer Katze und einem Affen. Zool.-bot. Ges., Wien, Verh., vol. 20, pp. 1027–1032.

GRATIOLET, L. P., AND P. H. E. ALIX 1866 Recherches sur l'anatomie du Troglodytes aubryi, chimpanzé d'une espéce nouvelle. Paris, Muséum d'histoire naturelle; Arch. du Muséum national d'histoire naturelle (Nouvelles Arch.), series 1, vol. 2, pp. 1–263.

GROSS, R. E. 1936 Congenital anomalies of the gallbladder. A review of 148 cases, with report of a double gallbladder. Arch. Surg., vol. 32, pp. 131–162.

HALPERT, B. 1927a Morphological studies on the gall bladder. I. A note on the development and the microscopic structure of the normal human gall bladder. Bull. J. Hopkins Hosp., vol. 40, pp. 390–408.

——— 1927b Morphological studies on the gall bladder. II. The "true Luschka ducts" and the "Rokitansky-Aschoff sinuses" of the human gall bladder. Bull. J. Hopkins Hosp., vol. 41, pp. 77–103.

HALPERT, B. 1929 The gall bladder. Its functions and some of their disturbances in the light of recent investigations. Arch. Surg., vol. 19, pp. 1037–1060.

HILT, L. M. 1937 Bilobed gall bladder (vesica fellea divisa). Radiology, vol. 28, pp. 233–234.

JAEGER (from Rau 1928 who ascribed the reference to Cullen, T. S. 1927, Brit. Med. J. I was unable to find this reference in the Brit. Med. J.).

KIRKMAN, H., AND R. E. STOWELL 1942 Renal filtration surface in the albino rat. Anat. Rec., vol. 82, pp. 373–391.

KREILKAMP, B. L., AND E. A. BOYDEN 1940 Variability in the composition of the sphincter of Oddi. A possible factor in the pathologic physiology of the biliary tract. Anat. Rec., vol. 76, pp. 485–497.

LEVYN, L., AND A. H. AARON 1926 Cholecystography by the oral method. Radiology, vol. 6, pp. 204–216.

LINEBACK, P. 1933 The respiratory, digestive and urinary systems. Chapter X. In Hartman and Straus, The anatomy of the rhesus monkey. Williams and Wilkins, Baltimore.

MANN, F. C., S. D. BRIMHALL AND J. P. FOSTER 1920 The extrahepatic biliary tract in common domestic and laboratory animals. Anat. Rec., vol. 18, pp. 47–66.

MAXIMOW, A. A., AND W. BLOOM 1942 A textbook of histology. Fourth edition. Saunders, Philadelphia.

MECKEL, J. F. 1838 Traité général d'anatomie comparée, vol. 8, Paris (trans. by Sanson and Schuster from the original, 1821, German edition).

MEYER, A. W. 1928 Doppelte Gallenblase. Deutsche Ztschr. f. Chir., vol. 211, pp. 404–405.

NUBOER, J. F. 1931 Studien über das extrahepatische Gallenwegssystem. Frankfurt. Ztschr. f. Path., vol. 41, pp. 198–249; 454–511.

OWEN, R. 1868 On the anatomy of vertebrates. London, vol. 3, p. 478.

PATZELT, V. 1936 Der Darm. IV. Die Darmabschnitte von besonderen Bau und der Darminhalt. 1. Der Magen-Darm-Übergang und des Duodenum. 2. Die Mündung der Leber und des Pankreas in den Darm. Hnd. der mikr. Anat. des Menschen, V (3) Verdauungsapparat III. Atmungsapparat, pp. 328–346.

PERRAULT, C. 1688 Memoir's for a natural history of animals. English translation, by A. Pitfeild, of original completed first French Edition (1676). (From Tyson 1699.)

PFUHL, W. 1932 Die Gallenblase und die extrahepatischen Gallengänge. Hndb. mikros. Anat. des Menschen V (2), Verdauungsapparat II, pp. 426–462.

PHILLIPS, M. H., D. ISAAC AND J. MACDONALD 1931 Four biliary anomalies: a plea for cholecystgastrostomy. Brit. Med. J., vol. 1, pp. 665–666.

POIRIER, P., AND A. CHARPY 1901 Traité d'anatomie Humaine. Deuxiéme édition, vol. 4. Annexes du tube digestif. Voies biliaires. Chapitre I. Anatomie, pp. 804–815 (Charpy). Chapitre II. Structure, pp. 784–803 (Soulié).

RAÚ, A. SUBBA 1928 Notes on a case of triplicate gall-bladder in Loris lydekkerianus Cabr. Half-yrly. J. Mysore Univ., vol. 2, pp. 10–19.

REX, H. 1888 Beiträge zur Morphologie der Säugerleber. Morph. Jb., vol. 14, pp. 517–617.

ROBINSON, B. 1907 Landmarks in the biliary and pancreatic ducts; physiological anatomical, and pathological. Med. Rec., N. Y., vol. 72, pp. 297–303.

RUCH, T. C. 1941 Bibliographia primatologica. Thomas, Springfield.

RUGE, G. 1902a Die äusseren Formverhältnisse der Leber bei den Primaten. Eine vergleichend-anatomische Untersuchung. Morph. Jb., vol. 29, pp. 450–552.

——— 1902b Ibid. III. Die Leber der platyrrhinen Westaffen. Morph. Jb., vol. 30, pp. 42–84.

——— 1906a Ibid. IV. Die Leber der Ostaffen (Simiae catarrhinae). Morph. Jb., vol. 35, pp. 75–239.

——— 1906b Ibid. V. Die Leber der Ostaffen (Simiae catarrhinae). Morph. Jb., vol. 36, pp. 93–275.

——— 1907 Ibid. VI. Die Leber des Menschen. Morph. Jb., vol. 37, pp. 397–487.

——— 1908 Ibid. VI. Die Leber des Menschen. (1. Fortsetzung.) Morph. Jb., vol. 37, pp. 614–660.

RUYSCH, F. 1721 Opera omnia Amstelodami observation XC, Vesicula Fellis bifurcata.

RYCROFT, B. W. 1923 A case of bilocular gall-bladder. Brit. J. Surg., vol. 10, pp. 579–580.

SANDIFORT 1840 Ontleedkundige beschouwing van een volwassen Orang-oetan. (S. Satyrus, Linn.) van het mannelijk geslacht. Verhand. Natuur. gesch. D. Nederl. overseesch. Bezittingen (Zool.), pp. 29–56 (from Flower, 1872).

SCHULTZ, A. H. 1933 Growth and development. Chapter II in Hartman and Straus, Anatomy of the rhesus monkey. Williams and Wilkins, Baltimore.

SCHWEGLER, R. A., AND E. A. BOYDEN 1937a The development of the pars intestinalis of the common bile duct in the human fetus, with special reference to the origin of the ampulla of Vater and the sphincter of Oddi. I. The involution of the ampulla. Anat. Rec., vol. 67, pp. 441–467.

——— 1937b Ibid. II. The early development of the musculus proprius. Anat. Rec., vol. 68, pp. 17–41.

——— 1937c Ibid. III. The composition of the musculus proprius. Anat. Rec., vol. 68, pp. 193–219.

SONNTAG, C. F. 1923 Anatomy, physiology and pathology of the chimpanzee. Proc. Zool. Soc. London, pp. 323–430.

——— 1924 On the anatomy, physiology, and pathology of the Orang-outan. Proc. Zool. Soc. London, pp. 349–450.

STOLKIND, E. 1940 Double gall-bladder. Report of a case and review of 38 cases. Brit. J. Surg., vol. 27, pp. 760–766.

STOWELL, R. E. 1941 Effect on tissue volume of various methods of fixation, dehydration, and embedding. Stain Tech., vol. 16, pp. 67–83.

STRACKER, O. 1909 Die Plica longitudinalis duodeni beim Menschen und bei Tieren. Sitzzsber. Akad. Wiss. Wien, Math.-Naturwiss. Kl., vol. 118, sec. IIb, pp. 375–437.

STRAUS, W. L., JR. 1936 The thoracic and abdominal viscera of primates, with special reference to the orang-outan. Proc. Amer. Phil. Soc., Philadelphia, vol. 76, pp. 1–85.

——— 1937 The visceral anatomy of an infant chimpanzee. J. Mammalogy, vol. 18, pp. 501–506.

SYMINGTON, J. 1890 On the viscera of a female chimpanzee. Proc. Royal Physical Soc. Edin., vol. 10, pp. 297–312.

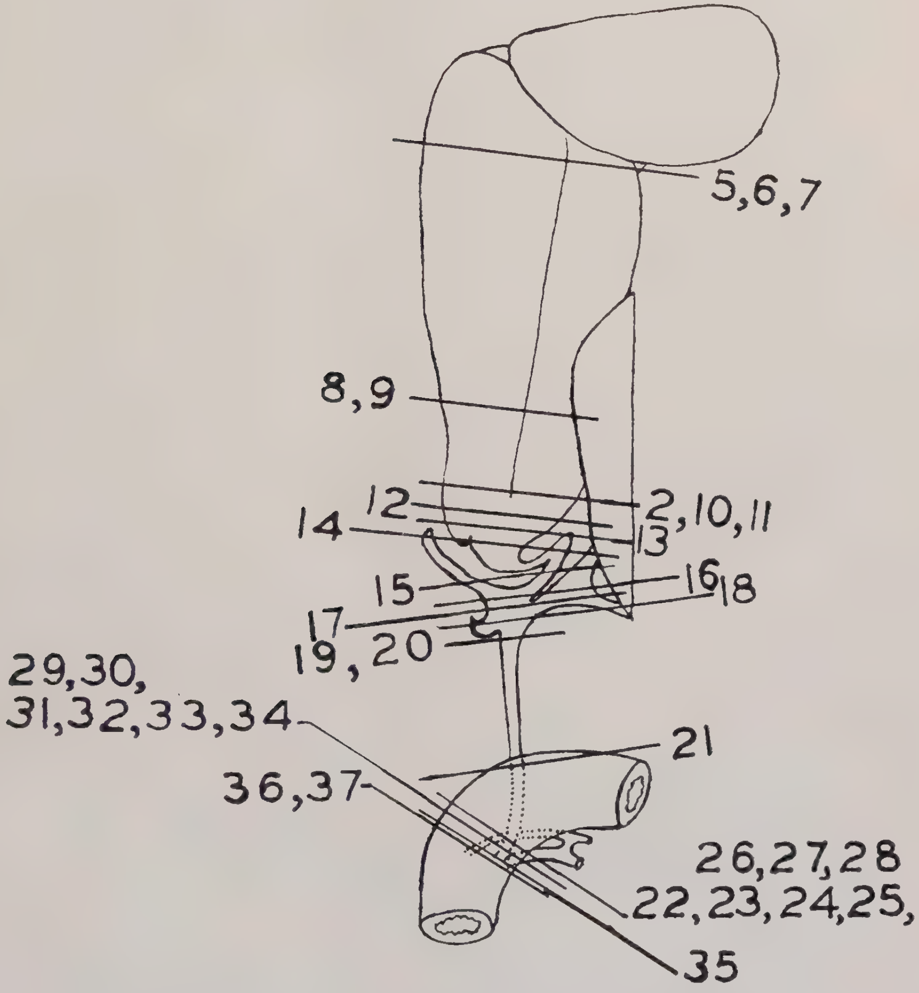
TITTLER, I. A. 1945 Two cases of accessory gall bladder in the rhesus monkey, Macaca mulatta. Anat. Rec., vol. 91, pp. 257–260.

TYSON, E. 1699 Orang-outang, sive Homo Sylvestris; or, the anatomy of a pygmie compared with that of a monkey, an ape, and a man. London, pp. 1–58.

WEISS, S. 1935 Diseases of the liver, gall bladder, ducts and pancreas: their diagnosis and treatment. Hoeber, N. Y.

ABBREVIATIONS

A, ampulla of Vater; APD, accessory pancreatic duct of Santorini; C, collum of gall bladder; CA, cystic artery; CC, common chamber formed by union of cystic duct and middle hepatic duct; CD, cystic duct; CDA, choledocho-duodenal aperture; CL, caudate lobe of liver; CLC, corpus of left compartment of bilobed gall bladder; CR, communicating ramus between accessory and main pancreatic ducts; CRC, corpus of right compartment of bilobed gall bladder; D, duodenum; DC, ductus choledochus; DG, duodenal glands of Brunner; E, esophagus; FL, falciform ligament; FVF, fossa vesica fellea; HA, hepatic artery; HS, hepatic (superior) surface of gall bladder; I, infundibulum of bilobed gall bladder; IS, isthmus connecting ''phrygian cap'' with corpus of right compartment; IVC, inferior vena cava; L, liver; LC, left compartment of bilobed gall bladder; LCL, left central lobe of liver; LH, ligamentum hepatoduodenale; LHD, left hepatic duct; LLL, left lateral lobe of liver; LM, longitudinal layer of duodenal muscularis; LN, lymph node; LP, lobulus paraventricularis of liver; LPV, left branch of portal vein; LS, area of liver serosa in direct contact with ''phrygian cap''; M, muscularis of gall bladder; MD, muscularis duodeni; MG, mucous glands; MHD, middle hepatic duct; MM, mucous membrane; MPD, main pancreatic duct of Wirsung; O, ostium of ampulla of Vater; P, pancreas; PC, ''phrygian cap''; PLD, plica longitudinalis duodeni; PM, peri-muscular layer of gall bladder; PP, papillary process of caudate lobe of liver; PV, portal vein; RC, right compartment of bilobed gall bladder; RCL, right central lobe of liver; RF, reinforcing fibers; RHD, right hepatic duct; RK, ''Rokitansky-Aschoff'' sinus; RLL, right lateral lobe of liver; RPV, right branch of portal vein; S, septum dividing gall bladder into two lobes; SA, sphincter ampullae; SC, sphincter choledochus; SE, serosa; SF, spiral fold of Heister; SFI, slight spiral elevations of mucosa in infundibulum of gall bladder; SP, sphincter pancreaticus; TP, tunica propria of mucosa; TS, tela submucosa.


5,6,7
8,9
12
14
2,10,11
13
15
16
18
17
19,20
29,30,
31,32,33,34
21
36,37
26,27,28
22,23,24,25,
35

4 Inferior surface of extra-hepatic biliary tract. The numbers refer to corresponding figures illustrating sections at the indicated levels. Magnification 2 ×.

PLATE 1

EXPLANATION OF FIGURES

All figures are of monkey no. 267 unless noted otherwise.

5 Inferior surface of ''phrygian cap,'' partially hidden by a portion of right central lobe of liver. Corpora sectioned at level indicated on figure 4. Magnification 1 ×.

6 As for figure 5, except that liver has been reflected from fundus.

7 View of isthmus connecting corpus of right compartment with the folded fundus. Blind end of left compartment shown also. Magnification 1 ×.

8 Transverse section through corpora at level indicated on figure 4. Mallory azan stain; 12 μ; magnification 2 ×.

9 Enlargement of portion of septum indicated on figure 8; magnification 30 ×.

10 Transverse section through infundibulum, near termination of septum; level indicated on figure 4. Verhoeff's elastic fiber stain; 14 μ; magnification 2 ×.

11 Enlargement of portion of left compartment near area indicated on figure 10. Mallory azan stain; 12 μ; magnification 30 ×.

12 Transverse section through infundibulum, just proximal to septum; level indicated on figure 4. Hematoxylin and eosin stain; 12 μ; magnification 2 ×.

13 Transverse section through proximal portion of the undivided infundibulum just distal to beginning of collum; level indicated on figure 4. To the left (apparent right) of the infundibulum is the terminal portion of the collum just before the start of the cystic duct. Magnification 2 ×.

14 Transverse section through beginning of collum at level indicated on figure 4. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

15 Section through left hepatic duct and termination of middle hepatic duct at levels indicated on figure 4. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

16 Common chamber formed by union of cystic and middle hepatic ducts. Left hepatic duct approaching union. Level indicated on figure 4. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

17 Section through termination of left hepatic duct in common chamber formed by union of cystic and middle hepatic ducts. Level indicated on figure 4. Foot's silver ammonium carbonate stain; 14 μ; magnification 2 ×.

18 Section through termination of right hepatic duct in common chamber. Level indicated on figure 4. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

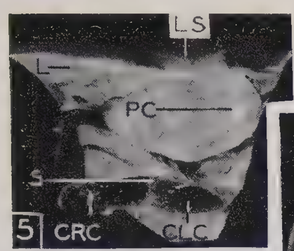
19 Transverse section through ductus choledochus at level of division of portal vein into right and left branches (see fig. 4). Mallory azan stain; 12 μ; magnification 2 ×.

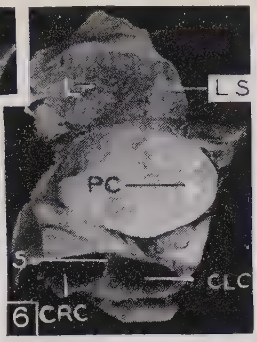
20 Part of wall of ductus choledochus from figure 19. Magnification 30 ×.

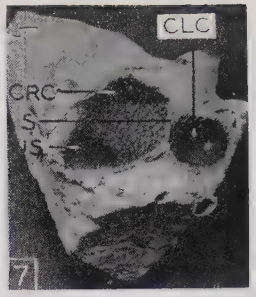
21 Transverse section through pancreatic portion of ductus choledochus at level indicated on figure 4; Mallory azan stain; 12 μ; magnification 2 ×.

22 Section through ductus choledochus and major pancreatic duct at level indicated on figure 4; sphincter choledochus well developed. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

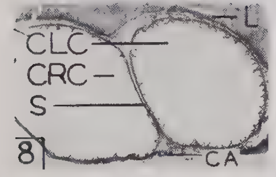
23 Section through ductus choledochus and sphincter choledochus at point of contact with major pancreatic duct. Approximate level indicated on figure 4. Mallory azan stain; 14 μ; magnification 2 ×.

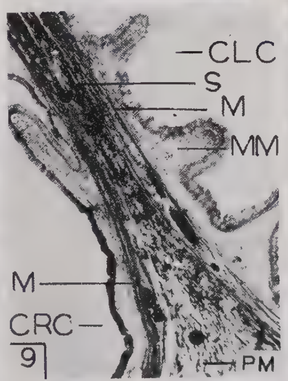

LS
L
PC
S
CRC
CLC
5

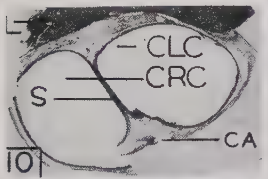

L
LS
PC
S
CLC
CRC
6

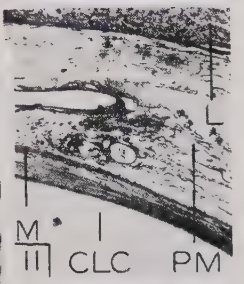


7

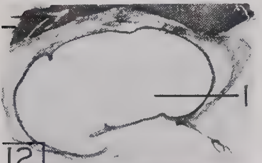

L
CLC
CRC
S
CA
8


CLC
S
M
MM
M
CRC
PM
9

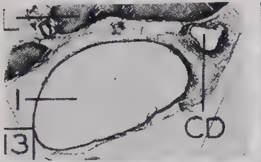

L
CLC
CRC
S
CA
10



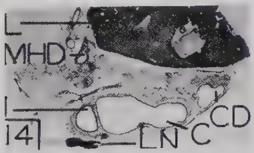
11

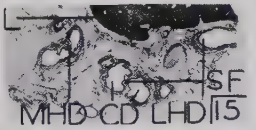


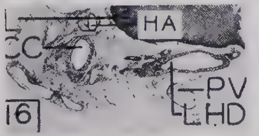
12

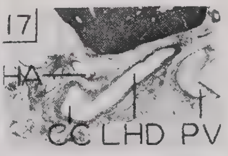


13

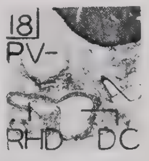

L
MHD
L
CD
LN
C
14


L
SF
MHD
CD
LHD
15

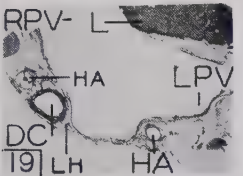

L
HA
CC
PV
L
HD
16

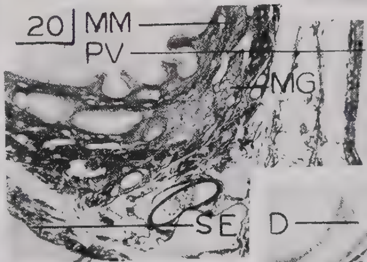


17

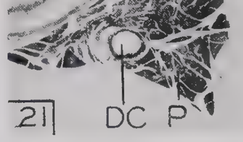


18

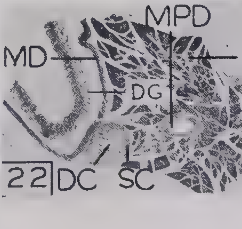

RPV
L
HA
LPV
DC
LH
HA
19

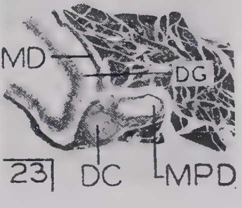


20



21


MPD
MD
P
DG
DC
SC
22



23

PLATE 2

EXPLANATION OF FIGURES

24 Detail of ductus choledochus and sphincter choledochus from figure 23. Magnification 14 ×.

25 ''Rokitansky-Aschoff'' sinus outside sphincter choledochus of figure 23. Magnification 30 ×.

26 Portion of sphincter of Oddi selected from figure 23. Magnification 30 ×.

27 Detail of dilated portion of major pancreatic duct from figure 23. Magnification 14 ×.

28 Ductus choledochus penetrating duodenal muscularis. Pancreatic duct in contact with ductus choledochus. Hematoxylin and eosin stain; 14 μ; magnification 2 ×.

29 Ductus choledochus and major pancreatic duct side by side in duodenal submucosa. Level indicated on figure 4. Mallory azan stain; 14 μ; magnification 2 ×.

30 Detail of ductus choledochus from figure 29. Magnification 16 ×.

31 Detail of major pancreatic duct from figure 29. Magnification 16 ×.

32 Ampulla of Vater at approximate level indicated on figure 4. Mallory azan stain; 14 μ; magnification 2 ×.

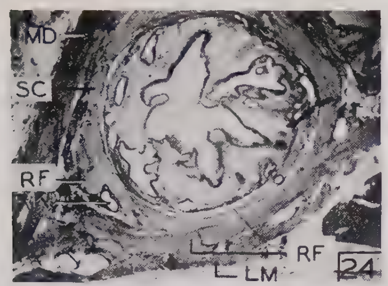
33 Ampulla of Vater at level indicated on figure 4. Foot's silver ammonium carbonate stain; 14 μ; magnification 2 ×.

34 Detail of figure 33. Magnification 14 ×.

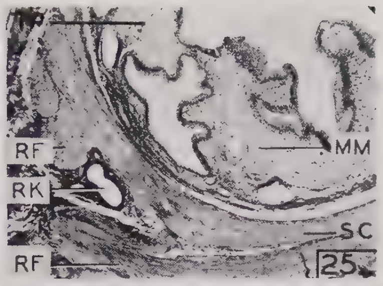
35 Ostium of ampulla of Vater. Level indicated on figure 4. Verhoeff's elastic fiber stain ;14 μ; magnification 4 ×.

36 Ostium of accessory pancreatic duct. Approximate level of section indicated on figure 4. Hematoxylin and eosin stain; 14 μ; magnification 4 ×.

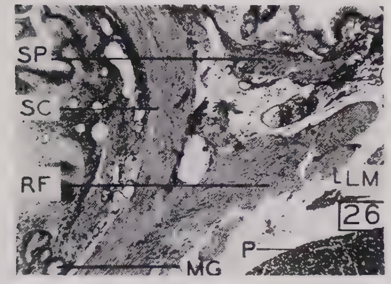
37 Accessory pancreatic duct in tunica muscularis duodeni. Mallory azan stain; 14 μ; magnification 4 ×.


MD
SC
RF
A
RF
LM

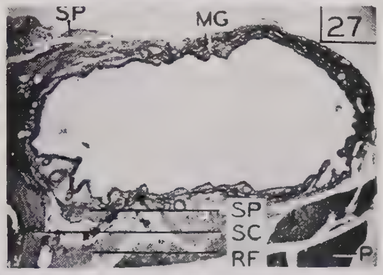
24


RF
RK
RF
MM
SC

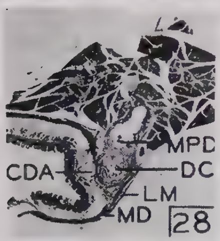
25


SP
SC
RF
LLM
P
MG

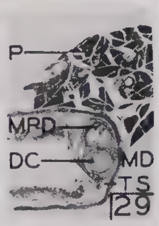
26


SP
MG
SP
SC
RF
P

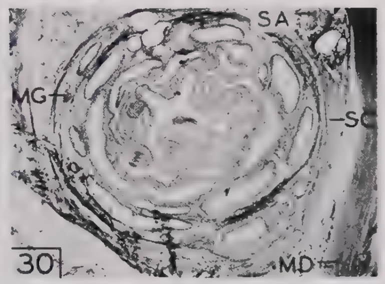
27


MPD
CDA
DC
LM
MD

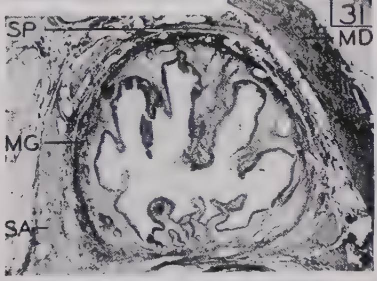
28


P
MPD
DC
MD
TS

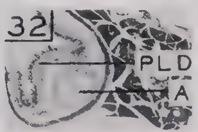
29



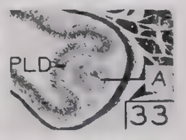
30



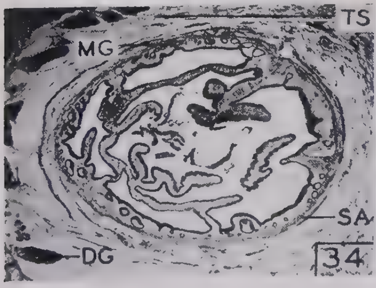
31



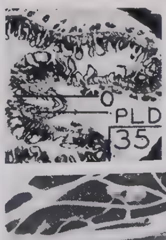
32



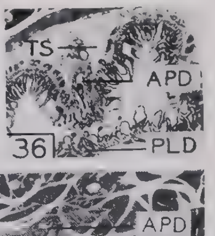
33



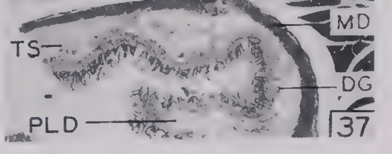
34



35



36


APD
MD
TS
DG
PLD

37

OLD AND "NEW" CONCEPTS ON THE STRUCTURE OF DENTIN

(A BIBLIOGRAPHIC NOTE)

V. O. HURME

Department of Operative Dentistry, College of Dentistry,
University of Illinois, Chicago

More than 100 years ago Alexander Nasmyth first presented the results of his studies on the minute anatomy of human teeth. While posterity has honored him as the discoverer of the cuticle which covers the enamel of a recently erupted tooth (1), it has remained almost completely ignorant of both the existence and the possible significance of his original contributions to the histology of dentinal tissue. However, instead of having to wait only 35 years for posthumous recognition, like Gregor Mendel, the father of modern genetics, Nasmyth has had to wait a full century for the first verification of his most important histologic researches (3, 4, 5).

Partial corroboration of Nasmyth's work has been provided by three Swiss investigators, Jaccard, Grosjean and Périer, whose contributions have all the more value since they were made without any apparent knowledge of the Scottish pioneer's labors during the first half of the nineteenth century. Although they published the details of their technic for the study of calcified tissues in 1940 and 1941 (7, 9) and even made special efforts to acquaint histologists outside Switzerland with the essential features of their methods (11), American anatomists as a group have not yet had an opportunity to become acquainted with their work. This may be due, at least in part, to the disruption of a normal exchange of scientific knowledge during the war years.

The information yielded by studying undecalcified teeth with the method developed in Switzerland threatens to upset most of the present-day concepts relating to the microscopic character of enamel and dentin. Since it is not possible, within the limits of a short article, to present a full review of the findings of the Swiss investigators, the writer wishes to limit his discussion to those phases of their work which appear to confirm certain observations made by Nasmyth a century earlier.

Through a curious chain of circumstances both anatomists and stomatologists have never paid much attention to Nasmyth's main work, which was given to the publisher by his widow shortly after his death in 1847. So far as the writer knows, Nasmyth's treatise is listed in only one of the few indices to dental literature, while none of the numerous histories of dentistry mention it. The listing in Weinberger's "Dental Bibliography" is not sufficiently clear to prevent confusing the identity of the work with an earlier publication bearing the same name; in fact, Prinz (12) erroneously refers to it as the second edition of the earlier publication. Equally baffling is the failure of the various authorities to refer to it; even W. D. Miller's extensive references to older dental literature reveal his unfamiliarity with the work. Since it is not likely that many readers today have access to the rare volume, it seems desirable to present Nasmyth's views on dentin by several direct quotations from his "Researches on the Developement, Structure, and Diseases of the Teeth" (5).

> "Ivory has been regarded as a structure composed of minute branching tubes, which radiate from the surface of the pulp . . . Moreover, the authors of this doctrine affirm that the material which connects and binds the tubes together, the intertubular substance, is amorphous and structureless."
>
> "The evidence . . . is the appearance which it presents when viewed with the microscope, the assumed injection of the tubes from the blood-vessels, the permeation of the tubes by ink, and the passage along them of bubbles of gas disengaged during the action of acids on the ivory . . . Such are the views which were entertained by physiologists relative to

the teeth up to the period when my own researches commenced . . . .''

''Again, it occurred to me that . . . the ivory is constantly subject to conditions which tend to wear away the surface, and consequently to expose the open extremities of the so-called tubes . . . Indeed it did appear to me most unaccountable that an organ like the tooth, subject to much violence and continued abrasion, should be furnished with tubes through which a constant transit of fluid was taking place, from the pulp towards the surface.''

''Again, Retzius and his followers remark that when a section of a tooth, made transversely to the direction of the fibres, is viewed upon a dark ground, a white spot is perceptible in the area of each of the so-called tubes. Now, this is the fact, but the inference to be drawn from it is directly opposed to that which Retzius endeavours to establish; for it is quite evident that if we view an object pierced by an aperture upon a dark ground, the area of the aperture, instead of being white, should look black, while the contrary proves that it is no hole at all.''

'' . . . Now, it appeared to me that if they were indeed tubes, they would be liable to collapse when their hardening calcareous material was withdrawn. But I have never seen them in a collapsed condition . . . Again, I have placed portions of this . . . in the field of the microscope, and subjected it to pressure under a magnifying power . . . , but I could never succeed in flattening the fibres in the manner that would occur had they been tubes, although the pressure which I employed was sufficient to crush ivory . . . ''

''It was quite evident to me, from the examination of preparations made [by pounding a tooth in a mortar until it was reduced to a coarse powder], that the so-called tube was in reality a solid fibre, composed of a series of little masses succeeding each other in a linear direction, like so many beads collected on a string . . . '' Nasmyth had asserted in 1839 that ''the animal residue consists of solid fibres, and if the decomposition [with 3% muriatic acid] be allowed to continue, these fibres present a peculiar baccated appearance . . . ''

'' . . . If the tissue of the ivory were composed of a series of tubes, with necessarily shut extremities, there would be no possibility of the entrance of injection into them, either of coloured or uncoloured fluid. It is moreover quite evident that so soon as the tissue had lost its natural moisture, these tubes would become filled with air, and the air having no means of exit, fluid of any kind could not be made to enter them. Taking the view of the structure of the tissue which I have pointed out, the possibility of fluid permeating the whole mass is quite admissible from its general porous nature . . . ''

Nasmyth contended that ''certain of the appearances [of dentin] were consequent upon the manner in which the preparations had been made'' and that under the influence of friction ''the appearance of a tube results from the hollowing out of the fibrous portion, while the surrounding [less calcified] interfibrous matter maintains a higher level.'' He preferred the term ''ivory'' as the best designation for the tissue and objected most strenuously to the term ''dentine,'' which, in his opinion, was ''not only an uncalled for addition to our already overcharged scientific nomenclature, but that it conveys, by its terminal syllable, the idea of the substance referred to being an elementary principle, which is not the case.'' He classified human dentin as the fibro-cellular variety of the tissue and did not hesitate to say that credit for discovering and propounding the concept of the cellular framework of the tissue belonged to him. He also maintained that that ''the fibres of the ivory must . . . be regarded as not only endowed with the functions of absorption, nutrition, and secretion, but also with that of sensation . . . ''

Nasmyth's concept of the microscopic structure of dentin was thus completely out of harmony with the views held by all other authorities before and after him. Van Leeuwenhoek had demonstrated dentinal canaliculi as early as 1678, Purkinje had seen them before 1835 and Retzius had published an elaborate discussion on them in 1837. The differences of opinion between the authorities never concerned the existence of the canaliculi and fibrils, but only certain details of structure, such as the size of the tubules, the existence of Neumann's sheath, the existence of anastomoses between the main tubules, etc. Even if Nasmyth's views had been known to dental histologists, it is not likely that they would have been favorably received.

The technic developed by Grosjean, Périer and Jaccard during 1939–1941 is similar to that employed by Nasmyth in that it does not require a decalcification of the tooth substance as a step for examination under the microscope (6, 7, 9). These authors claim that the processes of decalcification result in marked physico-chemical transformations of fine structural details, many of which may become completely destroyed before the tissue is ready for study (11). Not only that, but the histologic technics ordinarily used are likely also to produce various artifacts, which may be mistaken for authentic structural details later. The enamel spindles and tufts, they claim, are examples of such artifacts (8).

The Swiss technic is essentially an adaptation of a metallographic technic developed for the study of opaque solids (9, 11). The calcified tissue to be examined is cut with a saw or ground down with a carborundum wheel so as to present a plane surface, which is then cautiously smoothed with various abrasives and polished. The final polishing is done with pulverized aluminum of extreme fineness. In order to bring out certain structural details, the surface may be etched with a very weak acid for 1 to 10 seconds (never more!), or stained with a suitable dye in an aqueous solution, which is either neutral or slightly acid. The tissue so prepared is then examined or photographed with the aid of reflected light, which is obtained by using an illuminator of the type designed by Le Chatelier.

By preparing samples of human dentin in this manner, Jaccard and Grosjean have failed to discover any tubules in the tissue (10, 11). Longitudinal axial sections show rods with calcified contents, which are easily attacked by very dilute acids, or stained dark by various coloring agents. These rods have walls of an organic and highly acid-resistant composition, which are more resistant to polishing than the centers of the rods. Near the enamel and the cementum the dentin is formed of rods which are so closely packed together that it is difficult, except in perfect preparations, to see anything else than tissue of an irregular and chaotic appearance.

The dimensions of the rods are variable near the periphery, where they are only 3 to 4 μ in diameter, but those near the pulp chamber range from 5 to 8 μ. The number of the rods is larger peripherally than near the pulp chamber, as there is much branching of individual columns.

A transversely cut section gives a picture strikingly similar to a transverse section of enamel. The dentinal rods appear to be more or less hexagonal and they form a network which resembles a cross section of a honeycomb. This network is quite regular near the pulp chamber, but becomes less so toward the periphery due to the inclusion of many supplementary rods. In general, in sound teeth, the rods are solid and full, while sections cut from imperfectly calcified teeth may show lacunae in the rod contents. The last-mentioned observation by Jaccard and Grosjean was also anticipated by Nasmyth, who wrote that "this display, to which we are indebted for a demonstration of the original composition of the fibre, is evidently a partial arrest of development; the tissue being in its most perfect state when so closely aggregated that the compartments are not discernible."

The Swiss reports discussed in this paper, while denying the existence of tubuli in human dentin, make no mention of any special structures in the central portions of the dentinal rods. Nasmyth, on the other hand, recognized minute rounded bodies in these areas and shows them in some of his diagrams. He thought that they were the nuclei of the "ossified" cells which form the dentinal fibers (or rods). In the "Three Memoirs" (3) published in 1841 he wrote as follows about their microscopic appearance: "Generally, these [recently ossified] cells form a regular and complete coating, studded with points, which are placed at intervals, corresponding in extent to those between the fibres . . . These points are rendered visible from the greater opacity of the intermediate material, and will be seen to reflect or absorb the light, according to the difference in the focal distance." Jaccard and Grosjean recorded the observation that while the centers of transversely cut dentinal rods appeared undifferentiated from the rest of the rod contents when examined by reflected light, they appeared dark — as Tomes' fibrils — in tissue illuminated by transmitted light (11).

LITERATURE CITED

The literature consulted in the preparation of this article makes it desirable to list in full the original sources of information. A few explanatory notes have been added. The listing is arranged in chronological sequence.

1. Nasmyth, A.: On the structure, physiology, and pathology, of the persistent capsular investments and pulp of the tooth. *Medico-chirurgical Transactions* published by the Royal Medical and Chirurgical Society of London, 22: 310–328, January 22, 1839.

2. Nasmyth, A.: Researches on the Developement, Structure, and Diseases of the Teeth. London: John Churchill, 1839. (This volume contains only a Historical Introduction.)

3. Nasmyth, A.: Three Memoirs on the Developement and Structure of the Teeth and Epithelium. London: John Churchill, 1841. (Contains reproductions of Nasmyth's original diagrams, in color, and interesting commentaries on the antagonism which existed between the author and Richard Owen.)

4. Nasmyth, A.: Researches on the Developement, Structure and Diseases of the Teeth. Baltimore: Woods and Crane, Printers, 1842. (This volume is not an exact duplication of the second work listed in this bibliography, but contains numerous later comments on the character of dentin. It was published by the American Society of Dental Surgeons as Volume I of the American Library of Dental Science.)

5. Nasmyth, A.: Researches on the Developement, Structure, and Diseases of the Teeth. London: John Churchill, 1849. (Although bearing precisely the same title as the volume containing the historical introduction, which appeared in 1839, this work is not identical with it.)

6. Grosjean, M., and Périer, A.: Application des procédés optiques métallographiques à l'étude microscopique des tissus durs (dents et os). Compte Rendu des Séances de la Société de Physique et l'Histoire Naturelle de Genève (Supplement aux Archives des Sciences Physiques et Naturelles, 1939.), 56: 88–89, April-July, 1939. (This reference is incorrectly given in some papers in Swiss journals.)

7. Grosjean, M., Jaccard, R., and Périer, A.: Recherches sur la structure histologique de la dent par examen microscopique en lumière réfléchie. Schweizerische Monatsschrift für Zahnheilkunde, 50: 229–237, March, 1940.

8. Jaccard, R., and Grosjean, M.: Structure de l'émail dentaire: Quelques remarques basées sur la méthode d'observation ''surfaces polies et lumière réfléchie.'' Schweiz. Monatsschr. f. Zahnh., 51: 190–217, March, 1941.

9. Grosjean, M., and Jaccard, R.: Technique utilisée pour l'étude en lumière réfléchie des tissus durs (os et dents). Schweiz. Monatsschr. f. Zahnh., 51, 911–917, Nov., 1941.

10. Jaccard, R., and Grosjean, M.: Structure de la dentine. Schweiz. Monatsschr. f. Zahnh., 52: 335–347, April, 1942.

11. Jaccard, R., and Grosjean, M.: Comparison des méthodes employées pour l'examen des tissus durs (os et dents). Schweiz. Monatsschr. f. Zahnh., 52: 855–871, Oct., 1942.

12. Prinz, H.: Dental Chronology. Philadelphia: Lea and Febiger, 1945.

THE ARTERIAL SUPPLY OF THE VERMIFORM APPENDIX

M. A. SHAH AND MUBARIKA SHAH
Department of Anatomy, Dow Medical College, Karachi, India

ONE FIGURE

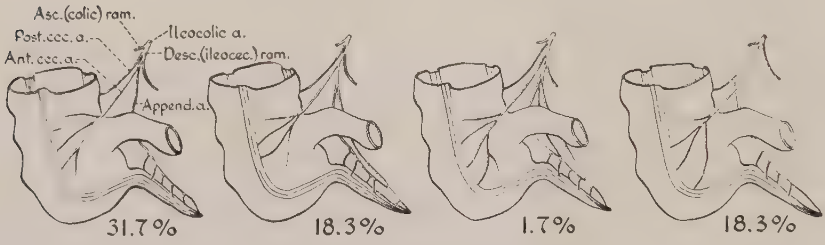
It is a matter of common knowledge, gained primarily from the current text books on Anatomy (Johnston and Willis, '42; Frazer, '37; Brash and Jamieson, '40) that the vermiform appendix is supplied by one artery, a twig from the descending (ileo-cecal) branch of the ileo-colic artery, which is said to be its sole arterial supply (Grant, '38). The other branches of the ileo-cecal are described as going to the anterior and posterior surfaces of the cecum (anterior and posterior cecal arteries) and to the terminal 5 or 6 inches of the ileum (ileal branch). Similarly, text books on Surgical Anatomy (Beesley and Johnston, '39; Rogers, '44; Lee McGregor, '43), and on Surgery (Thomson and Miles, '30) also speak of one appendicular artery.

During routine dissections of the cadaver, instances were noticed where the appendix appeared to have a dual blood supply. This fact was further investigated by careful dissections in sixty adult injected bodies used for dissection by the students in the dissection hall of the King Edward Medical College, Lahore, and the Dow Medical College, Hyderabad, Sind (now in Karachi). The coloring material used in injections was red lead.

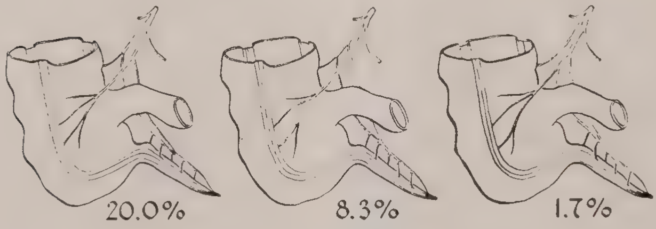
It was found that out of sixty bodies forty-two (70% had single appendicular arteries (upper row, fig. 1) and that eighteen (30%) had more than one artery (lower row, fig. 1).

As recorded in the upper row of figures, it will be seen that an appendicular artery answering to the typical book description was met with in nineteen specimens (31.7%), that in another eleven (18.3%) the artery, although arising as a single stem from the anterior cecal, divided into two distinct branches before entering the meso-appendix; and that in one instance (1.7%) it divided into three branches. In the remaining eleven specimens (18.3%) the artery was a branch of the posterior cecal, which is not surprising in view of the fact that the appendix, being an erstwhile portion of the cecum, may, in some cases, derive its supply from the cecal blood vessels. It is to be noted that whenever the posterior cecal artery supplied the appendix, either totally or partially, it always supplied the part nearest the base.

Fig. 1 — Variations in the arterial supply to the appendix. (Explanation in the text)



Single appendicular arteries



Double appendicular arteries

The lower row of figures represents the eighteen specimens in which there was more than one appendicular artery. In twelve instances (20.0%) one branch to the appendix was given off from the anterior cecal artery and another from the posterior cecal (the latter supplying an area starting at the base and extending for a variable distance along the length of the appendix). The branch from the posterior cecal is not to be confused with a small twig usually given off by the posterior cecal to the root of the appendix (Massie, '44). In another five specimens (8.3%) two appendicular arteries arose from the posterior cecal artery, and in one (1.7%) two appendicular arteries arose from the anterior cecal artery.

SUMMARY

Data are presented on the arterial supply to the vermiform appendix, based upon dissections of sixty adult cadavers. It is pointed out that, contrary to general impressions, there is some range of variation in the arterial supply to the appendix and that in 30% of cases the appendix receives two branches from either the anterior or the posterior cecal artery, or one branch from each of these.

ACKNOWLEDGMENTS

One of us (M.A.S.) is indebted to Prof. M. A. H. Siddiqui, Head of the Department of Anatomy, King Edward Medical College, Lahore, for some of the material obtained while working in that institution. Thanks are also due to the Anatomy students working in that institution during the years 1943 and 1944 for their ready co-operation.

LITERATURE CITED

Beesley, and T. B. Johnston 1939 Quoted by T. Seshachlan and S. R. Gorur, Ind. Medl. Gaz. L. V. P., p. 693, Oxford University Press, London.

Brash, J. C., and E. B. Jamieson 1940 Cunningham's Manual of Practical Anatomy, p. 305. Oxford University Press, London.

Frazer, J. E. 1937 Buchanan's Manual of Anatomy, p. 797. Bailliere, Tindall and Cox, London.

Grant, J. C. B. 1938 A Method of Anatomy, p. 160 Williams and Wilkins, Baltimore, Md.

Johnston, T. B, and J. Willis 1942 Gray's Anatomy, Descriptive and Applied, p. 766. Longmans, Green and Co., London.

Lee McGregor, A. 1943 A Synopsis of Surgical Anatomy, p. 46. John Wright and Sons, Bristol.

MASSIE, G. 1944 Surgical Anatomy, p. 235. Lea and Febiger, Philadelphia.

ROGERS, L. 1944 Treves Surgical Applied Anatomy, p. 467. Cassels and Co., London.

THOMSON, AND A. MILES 1930 Quoted by T. Seshachlan and S. R. Gorur, loc. cit., Oxford University Press, London.

AMERICAN SOCIETY OF ZOOLOGISTS

Officers for 1946

President	D. E. Minnich
Vice-President	E. G. Butler
Secretary	L. V. Domm
Treasurer	A. C. Kinsey

Executive Committee

(In addition to the four officers)

R. E. Coker	to serve until December, 1946
L. L. Woodruff	to serve until December, 1947
T. S. Painter	to serve until December, 1948
Sewall Wright	to serve until December, 1949
A. S. Pearse	to serve until December, 1950

Representative of the Society in the Division of Biology and Agriculture of the National Research Council

To serve until July 1, 1947 . . . . C. W. Metz

D. M. Whitaker, *Alternate*

Representatives of the Society on the Council of the American Association for the Advancement of Science

A. Elizabeth Adams
Waldo L. Schmitt

Representatives of the Society on the Council of the Union of American Biological Societies

H. B. Adelmann
H. H. Plough

Representatives of the Society on the Advisory Editorial Board of Biological Abstracts

M. Demerec
R. K. Enders
R. T. Kempton
E. J. Lund

Representative on the Executive Committee of the Biological Stain Commission

S. I. Kornhauser

Managing Editor of the Journal of Morphology

To serve until December, 1951 . . . . E. G. Butler

Members of the Editorial Board of the Journal of Morphology

To serve until December, 1946

H. B. Baker
Hope Hibbard
R. L. King

To serve until December, 1947

R. K. Burns
Mary J. Guthrie
A. S. Romer

To serve until December, 1948

G. Fankhauser
T. M. Sonneborn
D. M. Whitaker

LIST OF FORMER OFFICERS

American Morphological Society

President	*Vice-President*	*Secretary-Treasurer*
1890—E. B. Wilson	. . . .	J. P. McMurrich
1891—C. O. Whitman	E. L. Mark	J. P. McMurrich
1892—C. O. Whitman	H. F. Osborn	J. P. McMurrich
1893—C. O. Whitman	E. B. Wilson	J. P. McMurrich
1894—C. O. Whitman	W. B. Scott	G. H. Parker
1895—E. B. Wilson	W. B. Scott	G. H. Parker
1896—E. L. Mark	H. F. Osborn	G. H. Parker
1897—C. S. Minot	S. I. Smith	G. H. Parker
1898—H. F. Osborn	T. H. Morgan	G. H. Parker
1899—E. G. Conklin	W. M. Wheeler	Bashford Dean
1900—T. H. Morgan	H. C. Bumpus	J. S. Kingsley
1901—J. S. Kingsley	E. A. Andrews	T. H. Montgomery
1902—H. C. Bumpus	G. H. Parker	M. M. Metcalf

Additional Members of the Executive Committee

1891—E. B. Wilson
H. F. Osborn

1892—E. L. Mark
T. H. Morgan

1893—T. H. Morgan
C. B. Davenport

1894—E. A. Andrews
F. H. Herrick

1895—T. H. Morgan
S. Watase

1896—E. G. Conklin
William Patten

1897—J. S. Kingsley
Bashford Dean

1898—C. B. Davenport
F. R. Lillie

1899—J. P. McMurrich
G. H. Parker

1900—F. R. Lillie
Jacob Reighard

1901—C. F. W. McClure
C. W. Hargitt

1902—H. S. Jennings
R. G. Harrison

American Society of Zoologists

EASTERN BRANCH	*President*	CENTRAL BRANCH
G. H. Parker	1903	Jacob Reighard
E. A. Andrews	1904	C. H. Eigenmann
W. E. Castle	1905	F. R. Lillie
W. E. Castle	1906	C. C. Nutting
C. B. Davenport	1907	S. A. Forbes
W. M. Wheeler	1908	E. A. Birge
H. S. Jennings	1909	E. A. Birge
T. H. Montgomery	1910	C. E. McClung
H. V. Wilson	1911	George Lefevre
A. G. Mayer	1912	H. B. Ward
Raymond Pearl	1913	H. B. Ward

LIST OF FORMER OFFICERS

EASTERN BRANCH	*Vice-President*	CENTRAL BRANCH
Jacob Reighard	1903	H. F. Nachtrieb
W. E. Castle	1904	S. J. Holmes
William Patten	1905	William A. Locy
William Patten	1906	George Lefevre
F. H. Herrick	1907	H. B. Ward
H. S. Jennings	1908	M. F. Guyer
H. V. Wilson	1909	M. F. Guyer
H. H. Wilder	1910	H. F. Nachtrieb
H. E. Crampton	1911	R. H. Walcott
G. A. Drew	1912	C. M. Child
Alex. Petrunkevitch	1913	C. M. Child

Secretary-Treasurer

G. A. Drew	1903	Frank Smith
G. A. Drew	1904	F. R. Lillie
H. S. Pratt	1905	C. E. McClung
H. S. Pratt	1906	T. G. Lee
C. J. Herrick	1907	T. G. Lee
L. L. Woodruff	1908	T. G. Lee
L. L. Woodruff	1909	Charles Zeleny
H. W. Rand	1910	H. V. Neal
Raymond Pearl	1911	H. V. Neal
J. H. Gerould	1912	W. C. Curtis
Caswell Grave	1913	W. C. Curtis

Executive Committeemen

F. R. Lillie	Herbert Osborn
T. H. Montgomery	C. H. Eigenmann
H. C. Bumpus	J. G. Needham
H. S. Jennings	S. J. Holmes
E. A. Andrews	W. A. Locy
W. R. Coe	C. M. Child
G. A. Drew	R. H. Walcott
M. M. Metcalf	W. C. Curtis
D. H. Tennent	Oscar Riddle
R. G. Harrison	H. B. Ward
H. E. Jordan	Chauncey Juday
C. E. McClung	H. W. Norris
George Lefevre	C. E. McClung
T. G. Lee	H. F. Nachtrieb

AMERICAN SOCIETY OF ZOOLOGISTS (AMALGAMATED)

	President	*Vice-President*	*Executive Committeemen*
1914.	C. E. McClung	M. F. Guyer	H. E. Jordan—1 year
1915.	W. A. Locy	W. E. Ritter	H. F. Nachtrieb—2 years
1916.	D. H. Tennent	Charles Zeleny	H. V. Wilson—3 years
1917.	M. M. Metcalf	Charles Zeleny	George Lefevre—4 years
1918.	George Lefevre	L. L. Woodruff	A. F. Shull—5 years
1919.	C. M. Child	H. H. Wilder	D. H. Tennent—5 years
1920.	Gilman A. Drew	Caswell Grave	R. P. Bigelow—5 years
1921.	Charles A. Kofoid	Aaron T. Treadwell	L. J. Cole—4 years
1922.	H. H. Wilder	Bennet M. Allen	H. V. Wilson—5 years
1923.	M. F. Guyer	Robert A. Budington	M. M. Metcalf—5 years
1924.	R. G. Harrison	R. K. Nabours	George Lefevre—5 years
1925.	C. R. Stockard	C. R. Moore	C. M. Child—5 years
1926.	S. O. Mast	W. C. Allee	G. A. Drew—5 years
1927.	S. J. Holmes	Robert Chambers, Jr.	C. A. Kofoid—5 years
			H. H. Wilder—5 years
			J. H. Gerould—1 year
1928.	Caswell Grave	M. H. Jacobs	M. F. Guyer—5 years
1929.	C. B. Davenport	Fernandus Payne	R. G. Harrison—5 years
1930.	H. V. Neal	E. E. Just	C. R. Stockard—5 years
1931.	Fernandus Payne	D. E. Minnich	S. O. Mast—5 years
1932.	W. C. Curtis	L. V. Heilbrunn	S. J. Holmes—5 years
1933.	Charles Zeleny	J. H. Bodine	Caswell Grave—5 years
1934.	A. H. Sturtevant	H. W. Rand	C. B. Davenport—4 years
1935.	R. W. Hegner	Sewall Wright	H. V. Neal—5 years
1936.	W. C. Allee	C. W. Metz	Fernandus Payne—5 years
1937.	F. L. Hisaw	Helen D. King	W. C. Curtis—5 years
1938.	M. H. Jacobs	T. S. Painter	Charles Zeleny—5 years
1939.	J. T. Patterson	H. B. Goodrich	A. H. Sturtevant—5 years
1940.	W. R. Coe	D. H. Wenrich	Robert W. Hegner—5 years
1941.	R. E. Coker	J. P. Visscher	W. C. Allee—5 years
1942.	L. L. Woodruff	C. G. Hartman	F. L. Hisaw—5 years
1943.	T. S. Painter	L. H. Snyder	M. H. Jacobs—5 years
1944.	Sewall Wright	H. W. Stunkard	J. T. Patterson—5 years
1945.	A. S. Pearse	J. W. Gowen	W. R. Coe—5 years

Secretary-Treasurer

1914–1918.	Caswell Grave	1918–1921.	W. C. Allee

	Secretary		*Treasurer*
1921–1924.	W. C. Allee	1922	D. H. Tennent
1925–1928.	D. E. Minnich	1923–1924.	R. H. Bowen
1928–1929.	L. B. Arey	1925–1930.	L. B. Arey
1929–1930.	D. E. Minnich	1930–1933.	A. B. Dawson
1930–1933.	W. H. Cole	1933–1935.	B. H. Willier
1933–1936.	H. B. Goodrich	1935–1938.	W. N. Hess
1936–1939.	E. G. Butler	1938–1941.	T. C. Nelson
		1941–1944.	H. W. Beams

Representatives in the Division of Biology and Agriculture, National Research Council

F. R. Lillie 1919–1923
G. H. Parker 1919–1922
M. F. Guyer 1919–1921
William Patten 1921–1924
H. S. Jennings 1922–1925
E. G. Conklin 1923–1926
J. T. Patterson 1924–1927
L. L. Woodruff 1925–1928
E. G. Conklin 1926–1929
G. H. Parker 1926–1929
H. S. Jennings 1929–1931
L. C. Dunn 1931–1932
J. H. Bodine 1932–1935
L. V. Heilbrunn 1935–1938
Laurence Irving 1938–1941
D. E. Minnich 1941–1944

Associate Editors of the Journal of Morphology

1921.	Gary N. Calkins	J. S. Kingsley	William Patten
1921–1922.	E. G. Conklin	M. F. Guyer	W. M. Wheeler
1921–1923.	C. A. Kofoid	F. R. Lillie	J. T. Patterson
1922–1924.	G. A. Drew	H. V. Neal	L. L. Woodruff
1923–1925.	Caswell Grave	D. H. Tennent	H. V. Wilson
1924–1926.	Helen Dean King	S. O. Mast	A. A. Schaeffer
1925–1927.	R. W. Hegner	Fernandus Payne	H. B. Ward
1926–1928.	L. J. Cole	J. H. Gerould	M. H. Jacobs
1927–1929.	W. C. Curtis	J. Percy Moore	A. F. Shull
1928–1930.	Edgar Allen	G. A. Baitsell	R. E. Coker
1929–1931.	S. R. Detwiler	R. S. Lillie	H. J. Muller
1930–1932.	W. R. Coe	E. N. Harvey	H. E. Jordan
1931–1933.	L. V. Heilbrunn	S. O. Mast	G. K. Noble
1932–1934.	D. E. Minnich	B. H. Willier	L. L. Woodruff
1933–1935.	L. R. Cleveland	F. L. Hisaw	Franz Schrader
1934–1936.	Charles Zeleny	J. F. Daniel	J. S. Nicholas
1935–1937.	D. H. Tennent	L. C. Dunn	C. G. Hartman
1936–1938.	Sally Hughes-Schrader	E. E. Just	D. H. Wenrich
1937–1939.	Leigh Hoadley	W. A. Kepner	J. Percy Moore
1938–1940.	J. H. Bodine	N. E. McIndoo	H. H. Plough
1939–1941.	J. E. Kindred	H. W. Rand	S. A. Matthews
1940–1942.	A. M. Banta	V. Hamburger	C. W. Metz
1941–1943.	E. G. Butler	W. N. Hess	W. A. Kepner
1942–1944.	L. H. Hyman	A. C. Kinsey	H. W. Rand
1943–1945.	L. B. Arey	A. B. Dawson	C. C. Speidel

LIST OF PLACES OF MEETING

American Morphological Society

1890—Boston
1891—Philadelphia
1892—Princeton
1893—New Haven
1894—Baltimore
1895—Philadelphia
1896—Boston
1897—Ithaca
1898—New York
1899—New Haven
1900—Baltimore
1901—Chicago
1902—Washington

Central Naturalists

1899—Chicago
1900—Chicago

Society of American Zoologists

1901—Chicago
1902—Washington

American Society of Zoologists

EASTERN BRANCH

1903—Philadelphia
1904—Philadelphia
1906—New York
1907—New Haven
1909—Boston
1910—Ithaca

JOINT MEETINGS

1905—Ann Arbor
1908—Baltimore
1911—Princeton
1912—Cleveland
1913—Philadelphia

CENTRAL BRANCH

1903—St. Louis
1905—(Mar.) Chicago
1907—(Mar.) Madison
1907—Chicago
1910—(Apr.) Iowa City
1910—Minneapolis
1912—(Apr.) Urbana

MEETING PLACES

1914—Philadelphia
1915—Columbus
1916—New York
1917—Minneapolis
1918—Baltimore
1919—St. Louis
1920—Chicago
1921—Toronto
1922—Boston
1923—Cincinnati
1924—Washington
1925—New Haven
1926—Philadelphia
1927—Nashville
1928—New York
1929—Des Moines
1930—Cleveland
1931—New Orleans
1932—Atlantic City
1933—Boston
1934—Pittsburgh
1935—Princeton
1936—Atlantic City
1937—Indianapolis
1938—Richmond
1939—Columbus
1940—Philadelphia
1941—Dallas
1942—New York (cancelled)
1943—No meeting held
1944—Cleveland (Sept.)
1945—St. Louis (Mar. 1946)

AMERICAN SOCIETY OF ZOOLOGISTS

CONSTITUTION

Article I

NAME AND OBJECT

Section 1. The Society shall be called the ''American Society of Zoologists.''

Sec. 2. The object of the Society shall be the association of workers in the field of zoology for the presentation and discussion of new or important facts and problems in that science and for the adoption of such measures as shall tend to the advancement of zoological science.

Article II

MEMBERSHIP

Section 1. The membership of the Society shall consist of two classes:

a. Associate membership open to all actively engaged in the field of zoology who have had training in zoology equivalent to that required for the doctorate degree.

b. Active membership open to those who hold the doctor's degree in zoology or who have done work equivalent thereto and who, in addition to the usual requirements for this degree, have published the results of substantial zoological research.

Sec. 2. Election to membership in the Society shall be upon recommendation of the Executive Committee.

Sec. 3. Associate members shall not hold office or vote, but may present papers before the Society without introduction. Associate members shall receive all literature issued by the Society, including the proceedings of the Society, and shall have the same privileges as active members in journal subscriptions.

Sec. 4. Associate members may be elected to active membership in the usual way when the research requirements of active membership have been met.

Article III

OFFICERS

Section 1. The officers of the Society shall be a President, a Vice-President, a Secretary, a Treasurer, and the members at large of the Executive Committee.

Sec. 2. The Executive Committee shall consist of the President, the Vice-President, the Secretary, the Treasurer, and five members elected from the Society at large. Of these five members, one shall be elected each year to serve five years. If any member at large shall be elected to any other office, a member at large shall be elected at once to serve the remainder of his term.

Sec. 3. These officers shall be elected by ballot at the annual meeting of the Society and their official terms shall commence with the close of the annual meeting, except that the Secretary and the Treasurer shall be elected triennially and shall serve for three years.

Sec. 4. The officers named in Section 1 shall discharge the duties usually assigned to their respective offices. (See by-laws.)

Sec. 5. Vacancies in the board of officers, occurring from any cause, may be filled by election by ballot at any meeting of the Society. A vacancy in either the Secretaryship or the Treasurership occurring in the interval of the meetings of the Society may be filled, until the next annual meeting, by appointment by the Executive Committee.

Sec. 6. At the annual meeting the President shall name a nominating committee consisting of three active members. It shall be the duty of this committee to nominate officers of the Society, including the permanent representatives which the Society is asked to name to various boards and councils. This committee shall make its nomination to the Secretary at least four months before the next annual meeting. Additional nominations for any office may be made in writing to the Secretary by any five members at any time previous to balloting.

ARTICLE IV

MEETING OF THE SOCIETY

Section 1. Unless previously determined by the Society, the time and place of the annual meeting shall be determined by the Executive Committee. Special meetings may be called and arranged for by the Executive Committee. Notices of such meetings shall be mailed to all members of the Society at least two weeks before the date set for the meeting.

Sec. 2. Sections of the Society may be organized in any locality by not less than ten members, for the purpose of holding meetings for the presentation of scientific papers. Such sections shall have the right to elect their own officers and also associate members; provided, however, that associate membership in any section shall not confer membership in the Society.

ARTICLE V

QUORUM

Twenty-five members shall constitute a quorum of the Society and four a quorum of the Executive Committee.

ARTICLE VI

CHANGES IN THE CONSTITUTION

Amendments to this Constitution may be adopted at any meeting of the Society upon approval of two-thirds of the members voting, subject to the following conditions:

a. The proposed amendment must be in writing and signed by at least five members of the Society.

b. This signed proposal must be in the hands of the Secretary at least one month before the meeting of the Society at which it is to be considered.

c. The Secretary shall mail copies of the proposed amendment to the members of the Society at least two weeks before the meeting.

d. Members unable to attend may submit their votes in writing to the Secretary.

BY-LAWS

ARTICLE I

DUES

Section. 1. The annual dues for active and associate members, unless remitted or changed by the vote of the Society, shall be two dollars.

Any member in good standing, having paid annual dues to the Society for not less than twenty-five years, who shall change his professional status, may apply to the Executive Committee for the remission of further dues. Applicants approved by the Executive Committee shall be exempt from further payment of dues, but such members shall be entitled to all the privileges of membership formerly enjoyed.

Sec. 2. Any member in arrears of dues for two years should be dropped by the Executive Committee. Upon payment of arrearage he shall be reinstated.

ARTICLE II

PRESIDENT

Section 1. The President shall preside at scientific sessions and at the business meetings.

Sec. 2. He shall also appoint the committees prescribed by the constitution and by-laws.

ARTICLE III

VICE-PRESIDENT

Section 1. The Vice-President shall preside at sessions designated by the President.

Sec. 2. He shall also assume the duties of the President in the latter's absence.

ARTICLE IV

SECRETARY

Section 1. The Secretary shall keep the records of the Society.

Sec. 2. He shall be responsible for all arrangements for the meetings, including the appointment of local committees.

Sec. 3. Whenever the proper officers of a number of related societies shall have a conference covering matters of mutual interest to the several societies, he shall act as the delegate or representative of this Society.

Sec. 4. He shall make such purchases and employ such assistance as will, in his judgment, expedite the business of the Society, and

Sec. 5. He shall be reimbursed out of the funds of the Society for expenses incurred in attending meetings of the Society.

ARTICLE V

TREASURER

Section 1. The Treasurer shall be in charge of the funds of the Society.

Sec. 2. At the annual business meeting of the Society he shall present a statement to date of the funds of the Society.

Sec. 3. He shall make such purchases and employ such assistance as will, in his judgment, expedite the business of the Society.

ARTICLE VI

AUDITING COMMITTEE

The President shall annually appoint an auditing committee of two, who shall audit and report upon the financial record and statement of the Treasurer at the meeting for which they were appointed.

ARTICLE VII

PROGRAM RULES

In matters relating to programs for annual meetings, the following rules shall be observed:

Section. 1. Titles and abstracts of papers to be presented by any method at the annual meeting must be sent to the Secretary in duplicate before the final date set by him. The length, form, and arrangement of the abstracts must comply with the requests of the Secretary in order to insure prompt publication by The Wistar Institute. The titles and abstracts of papers which do not conform to such requests or papers which are received late may be presented by title only.

Sec. 2. Titles shall be listed in groups according to subject matter, the groups to be determined by the Secretary.

Sec. 3. Whenever conditions require, the Secretary shall schedule two or more groups for the same hour and arrange the program to bring together papers on subjects of more general interest for meetings of the whole Society.

Sec. 4. Each member may have not more than fifteen minutes of the time of the Society at the annual meeting. This time may be used by him to present one or more papers personally or to introduce papers of non-members.

Sec. 5. All papers not read when called for as listed shall be placed at the end of the group list, and if not read when called for the second time, shall be read by title only.

Sec. 6. The titles of ‘‘introduced’’ papers may be listed in the groups after the titles of papers to be read by members.

PROCEEDINGS OF THE FORTY-SECOND ANNUAL MEETING OF THE AMERICAN SOCIETY OF ZOOLOGISTS

SAINT LOUIS UNIVERSITY AND WASHINGTON UNIVERSITY
SAINT LOUIS, MISSOURI
MARCH 28, 29, 30, 1946

PROGRAM

The American Society of Zoologists held its first post-war meeting in Saint Louis, Missouri, on March 28, 29, 30, 1946. Due to prevailing conditions, plans for a meeting in 1945 had been virtually abandoned but the sudden and unexpected end of the war in early August changed matters. However, the task of preparing for such a meeting before the close of the year was too formidable, consequently the 1945 meeting was postponed until the spring of 1946. The regular 1946 meeting will be held in December as in pre-war years.

The Saint Louis meeting was held in conjunction with Section F of the American Association for the Advancement of Science and in association with several other biological societies. With the exception of the demonstration program, which was held on Friday afternoon in Rebstock Hall on the campus of Washington University, all meetings for the presentation of papers and symposia were held in the Hotel Jefferson in downtown Saint Louis.

The Saint Louis meeting was not so large as the meetings immediately preceding the war but it was well attended and the general consensus among those in attendance was that it was a success. The total number of papers on the program scheduled to be presented in person was 100, of which 16 were presented by demonstration. There were 105 papers included in the program as ''read by title.'' According to reports from

Presiding Officers, 7 papers were omitted. In all, therefore, 93 papers were actually presented in person at the sessions of the Society in Saint Louis. Included in the foregoing figures are papers which were given in the various symposia sponsored by the Society.

All sessions of the Society were well attended and while no record of actual attendance was available, the indications were that the number of Zoologists assembled in Saint Louis compared favorably with the number assembled at some of the meetings of the Society shortly before the war.

The Biologists' Smoker was held on the evening of the first day of the meeting, Thursday, March 28th, in the Grand Lounge of the Municipal Auditorium. There was a good attendance and refreshments were again provided after having been omitted at the last two meetings.

The Annual Dinner of the American Society of Zoologists was held on Friday evening, March 29th, in the Crystal Room of the Hotel Jefferson with 280 persons in attendance. An address on ''The Little Researcher'' was given by Dr. Carl G. Hartman, Vice-President of the American Association for the Advancement of Science and Chairman of Section F.

BUSINESS MEETING

The forty-second annual business meeting opened at 12:10 P.M., on Friday, March 29th, President A. S. Pearse presiding.

The following business was transacted:

1. *The minutes of the last meeting.* The minutes of the last meeting were published in the April 1945 supplement to The Anatomical Record and distributed to all members of the Society.

It was voted to approve the minutes as printed.

2. *Memorial resolutions.* The Secretary reported that during the past year the Society had suffered the loss by death of 12 active members. These were Arthur Mangun Banta, Thomas Barbour, Henry Lane Bruner, Wayland Morgan Chester, Frank Blair Hanson, Clarence Erwin McClung,

Thomas Hunt Morgan, Edwin Eustace Reinke, Bertram Garner Smith, Francis Bertody Sumner, Herbert Eugene Walter, and Henry Baldwin Ward. The following resolutions were prepared:

ARTHUR M. BANTA

Arthur Mangun Banta, Professor of Biology Emeritus at Brown University, died on January 2, 1946 at the age of sixty-eight. Dr. Banta was born in Greenwood, Indiana, on December 31, 1877. He received his A.B. degree from the University of Indiana in 1903 and, the following year, received his A.M. from the same institution. After receiving his Ph.D. degree from Harvard University in 1907, he became Professor of Biology at Marietta College for 2 years. In 1909, he became resident investigator in the Station for Experimental Evolution of Carnegie Institute at Cold Spring Harbor and remained there for 20 years. From 1929 until the time of his retirement (June, 1945), he held the position of Professor of Biology at Brown University.

Dr. Banta's contributions to science are many. The primary pattern of his research work concerned the influence of environment on the development of inherited characters in animals, and the intense interest he possessed in his chosen field was easily communicated to his many students. He became a foremost authority on Cladocera, and it was in this group of crustaceans that he found ideal material for his fruitful experiments.

During his teaching years at Brown, he built for himself a loyal following of students, both through his distinction as a scientist and his kindly interest in their problems. He was never too busy to give his full and undivided attention to his associates, either college freshman or colleague, and his thoughtful and wise counsel will be remembered by all who knew him.

Dr. Banta was a man of impressive features and a distinguished appearance. He was neighborly and kindly, and the true distinction of his character was ever apparent.

One of the saddest aspects of the passing of this scientist and teacher is the fact that he retired from teaching earlier than most in order to devote his full time to the many phases of his research work, which he felt were unfinished. He was looking forward with real enthusiasm to the years ahead. But this expectation was cut short after but 5 months by the heart attack from which he never recovered.

The American Society of Zoologists and the world of science have lost a valued member.

GEORGE W. KIDDER

THOMAS BARBOUR

Thomas Barbour, Director of the Museum of Comparative Zoology and Alexander Agassiz Professor of Zoology at Harvard University, was born on August 19, 1884, and died in Boston on January 8, 1946. His technical scientific work lay in the field of herpetology, but his interests were far wider.

He was familiar with and interested in every field of natural history, particularly that of the tropics, and was cast in the tradition of the great field naturalists, a worthy successor to such men as von Humboldt and Wallace.

The pattern of his later career as zoologist, traveller, sportsman, lover of the tropics, and museum director, began to appear in childhood and even infancy. He had been to Europe and returned (tucked in a bureau drawer) before his first birthday. His first childhood memories were of the bison in the Central Park Zoo. He visited most of the European natural history museums when 8 years old; caught his first trout when 9; began natural history exploration in Florida and the Bahamas at 14; became an intimate friend and protégé of Ditmars and the other Curators of the newly established Bronx Park Zoo in New York at 15. He graduated from Harvard in 1906. His honeymoon, shortly after this, characteristically took the form of a trip round the world with herpetological exploration in the East Indies as its major feature.

Throughout his life he journeyed time and again to the tropics, particularly those of America; the islands and lands bordering the Caribbean formed the center of his interests, and the problems of animal life on islands were of absorbing interest to him. Yachting trips with his friend Allison Armour facilitated his work on many occasions. During the past decade, his winters were spent in field work in Florida and Cuba.

He was interested in the development of biological stations in the tropics. He was Custodian of the Atkins Institution of Harvard University at Soledad, Cuba. He was one of the founders of the field station at Barro Colorado in the Canal Zone, later named the Canal Zone Biological Area, and was for many years its executive officer.

Dr. Barbour's association with and interest in the Museum of Comparative Zoology at Harvard began with his undergraduate years; his graduate work was done at the museum; and with the attainment of his doctorate, he became, in 1910, Curator of Reptiles and Amphibians. In 1922, he was appointed a Professor of Zoology; but his interests were centered in the museum and his research activities, and apart from the supervision of graduate students, he took very little part in teaching activities. During his curatorship he not only enriched greatly the herpetological collection but added to the museum's holdings in many other fields. In 1927, he became Director of the museum.

Following decades of vigorous activity and major achievements under the Agassizs, the institution had entered upon a period of stagnation; symbolic of this is the fact that the building was still in the era of gas lighting. With his accession, a major revolution, carried out with his characteristic energy, took place. Numerous changes and additions were made in the staff; extensive changes were made in the building arrangements; the elevator shaft was filled two floors deep with worthless materials to be carted away from its foot by the truckload. For the remainder of his life, the welfare of the museum was his major interest.

He was a member and in many cases had been an officer of a variety of American scientific societies, including the National Academy of Sciences, the American Academy of Arts and Sciences, the American Philosophical Society, etc., and of a number of foreign societies as well, and a holder of honorary doctorates from Harvard, Havana, Dartmouth, and the University of Florida.

Dr. Barbour was a man of breadth and stature, literally so in his physical build, more so in his abounding energy, activity and enthusiasms, and in his breadth of interest in persons and in the entire world of life. In his death we can truly say that we have lost a big man.

Alfred S. Romer

HENRY LANE BRUNER

The death of Henry Lane Bruner March 17, 1945, marked the passing of another middle western zoologist whose early interest in natural science led to advanced study in our eastern and in German institutions.

Born of pioneer parentage in Illinois, January 10, 1861, he took advantage of the educational opportunities open to him and was graduated from Abingdon College with the A.B. degree in 1880. Then followed a year at Sheffield Scientific School of Yale College and four summers as Assistant to the United States Fish Commission at Woods Hole. During this period friendships were established with young men who became leaders in zoology, and there was crystalized also, an interest in zoological research. Within a few years he went to Germany where he became a student of Wiedersheim in the University of Freiburg. Here he was granted the Ph.D. degree in 1896.

Seven noteworthy research contributions were made in the fields of comparative anatomy and physiology of amphibians and reptiles as well as some articles of lesser importance. They showed the results of greatest care, accurate interpretation and clear expression. As a professor in a small college Dr. Bruner's time for research was limited. For one year, 1910–11, he was able to devote his entire time to investigation and writing as Harrison Research Fellow in Zoology at the University of Pennsylvania.

Dr. Bruner preferred life in a small college related to the Disciples of Christ church of which he was a member. In 1892 he became Professor of Biology and Geology in Butler College after having held similar positions in Abingdon and Eureka Colleges and in Drake University. He remained connected with Butler the rest of his life as Head of the Department of Zoology 1919–38, Director of Graduate Studies 1932–38, and after 1938 as Professor Emeritus of Zoology. Butler awarded him the honorary Doctor of Science degree in 1932.

Tall, slender, erect, and with dignified bearing Dr. Bruner was quiet and scholarly. Somewhat austere he was friendly in counsel. He stood firm in his decisions and his judgment was respected. His lectures were clear, concise, and given without waste of effort or words. The students who were encouraged to become zoologists are evidence of his success as a teacher. He never lost interest in the beginning student nor his early enthusiasm for teaching general zoology. This interest was shown in his manual "Laboratory Directions in College Zoology" (Macmillan, third edition, 1942) which is widely used in beginning courses.

In 1890 he married Carolyn Aumock who died in 1894. In 1897 he married Emma Pfeiffer whom he had met while a student in Freiburg. She, a son and a daughter, and a son by the first marriage survive.

Nathan E. Pearson

WAYLAND MORGAN CHESTER

Wayland Morgan Chester, Professor of Biology Emeritus at Colgate University, Hamilton, New York, died January 1945 at the age of 74. Dr. Chester was born in Noank, Connecticut, on March 10, 1870 and came to Colgate after graduating from Mystic Valley Institute in Connecticut. Graduating from Colgate in 1894, he immediately joined the faculty as Assistant in Geology and Natural History while studying for the masters degree which he received 2 years later. In 1898 he became an Instructor in Biology and 6 years later became Head of the Department, a post he held until he retired in 1938. He also was Assistant Curator of Colgate's Museum of Natural History.

As a teacher Dr. Chester was inspiring and thorough, giving unstintingly of himself and his substance for the welfare and scientific advancement of his students. As his teaching led into the age of specialization he was always abreast of the most recent advancements. His teachings were summarily interpreted in the light of his broad personal experience with natural history.

Dr. Chester worked at the Bermuda Biological Station in summers and was recognized for research on coral animals done there and at Harvard University where he studied in 1909–10. He published few papers but his investigative desires were maintained to the last. After retiring from active teaching he spent his winter sojourns on the Florida beaches in search of sponges for which he had a genuine interest. He was a Fellow of the American Association for the Advancement of Science and a member of Phi Beta Kappa, Beta Theta Pi, American Society of Zoologists, Biological Society of Washington, and the American Microscopical Society.

Raymond J. Myers

FRANK BLAIR HANSON

Frank Blair Hanson was born in Bloomington, Illinois, July 15, 1886. He graduated from the Bloomington High School in 1905. He was in Government service in Washington, D. C., from 1905 to 1909. He studied under a scholarship at George Washington University from 1909–1913 and received his A.B. in 1913. From 1913–1914 he was an assistant at the University of Illinois, and in 1914–1915 studied there under a fellowship, receiving the degree of A.M. in 1915. He was a traveling Fellow of the American University, 1915–1916. He received his Ph.D. from the American University in 1919, having completed his thesis while teaching at Washington University, St. Louis.

He served in Washington University as Instructor in Zoology from 1916–1918, as Assistant Professor from 1918–1920, as Associate Professor from 1920–1924, and as Professor from 1924–1933. During a leave of absence from Washington University, he served for the period July 1, 1930 to June 30, 1932 as assistant in the fellowship program in Europe of the Rockefeller Foundation. He became Assistant Director in the Natural Sciences division of The Rockefeller Foundation September 1, 1933; in 1936 he became Associate Director, in which capacity he served until the time of his death on July 21, 1945.

He was a member of the Phi Beta Kappa, Sigma Xi, and Phi Sigma honorary societies and of the following scientific societies: American Association for the Advancement of Science; American Society of Zoologists; American Society of

Naturalists; Genetics Society of America; American Association of Anatomists; Society for Experimental Biology and Medicine; and St. Louis Academy of Science.

He was married on June 21, 1910 to Harriet Roman Cavender of Maryland, who survives him. Besides his wife, he is survived by two children, Miss Blair Hanson, Assistant Professor of French at Allegheny College, Meadville, Pennsylvania; and Dr. Phyllis Claire Hanson, Assistant in Pathology, University of Rochester, New York. A son, Frank Blair Hanson, Jr., a graduate of Oberlin College, was a Lieutenant, Junior Grade, when he was lost with his ship, the destroyer escort Frederick C. Davis, sunk in the Atlantic by a German submarine a few days before VE day. This loss was naturally a great blow to Dr. Hanson and no doubt contributed to his ill health during the months that intervened before his death.

Dr. Hanson was the author of some fifty scientific papers. His earlier work was in comparative anatomy and embryology. His later work and major interest as an investigator was in genetics, including radiation genetics. Notable on his list of publications, were those concerning the effects of alcohol fumes and x-rays upon the albino rat and the problem of their inheritance, and those concerning x-rays and radium rays as effecting mutations and other changes in Drosophila. The importance of this later work was recognized by grants from the American Association for the Advancement of Science and from the Committee on Radiation of the National Research Council.

The writer of this obituary recalls his first meeting with Dr. Hanson about 1922 during a visit to the Department of Zoology at Washington University. In the absence of my old friend Caswell Grave, Dr. Hanson did the honors and among other things he showed me his rat colony and explained what he was doing and what he thought about the effects of the alcohol fumes upon his rats. I was interested in the problem and in the rats but I was more interested in the man, because I was so taken with the clarity of his statements regarding his work. I sensed the fact that here was a competent investigator and also a man of the highest intellectual integrity. One outcome of this contact and later ones was that Dr. Hanson was tendered, in 1923, a professorship at the University of Missouri to succeed Dr. George Lefevre who had died a few months earlier. It has been always a matter of deep regret to me that Dr. Hanson decided to remain at Washington University.

Our mutual exchanges at that time were the beginning of a relationship that made him one of my half dozen closest zoological friends during the remainder of his life. Later, we were both interested in work fostered by the Committee on Radiation of the National Research Council, and being often in St. Louis I was repeatedly a guest in his home. The children were in that delightful pre-high-school age, although I do not know that any age of childhood is more delightful than another, and I became well acquainted with the entire family. Their life together always seemed to me like a beautiful dream, and my intimacy with them continues. Hence, the loss of Dr. Hanson and of Frank Jr. was a very personal matter to me as I am sure his loss must be to many others who knew him intimately. Under such circumstances it is well-nigh impossible to say what one feels.

Frank Blair Hanson was, in my opinion, a zoologist of unusual ability and a man of the highest integrity. My impression that he was also a man of excellent judgment received ample confirmation when he was selected by the Rockefeller Foundation for appointment as Assistant Director, in 1933, and later as Associate Director of the Natural Sciences in that organization. I recall his great regret in 1933 when he was leaving for good, as he feared, his work as an investigator. But he had a family to educate and one very much worth educating, and his professor's salary at that time gave no hope that he could do for his children what he wished. We met often during his years with the Foundation and all my earlier impressions were confirmed. His career there removed a very competent investigator from the field of zoological research, but his conscientious and intelligent conduct of his responsibilities as an officer of the Foundation contributed to the advance of zoological science. By giving up his own research he helped make it possible for many other investigators to continue theirs with the aid of the foundation which he advised. There is perhaps no less credit when a man thus lays down his work, important though it be, and as a result enables his fellows to do theirs with greater effect. And Frank Blair Hanson did that.

W. C. CURTIS

CLARENCE ERWIN McCLUNG

Dr. Clarence Erwin McClung died very suddenly on January 17, 1946 in Swarthmore, Pennsylvania.

As is true of many men of accomplishment, Professor McClung had a rugged boyhood. He was born in a mining town, Clayton, California, April 5, 1870, his father being a civil and mining engineer and his mother the daughter of a physician. His elementary schooling suffered as his family moved about from place to place and it was not until high school age that he was able to attend public schools regularly. He graduated from the School of Pharmacy at Kansas University, receiving the Ph.G. degree in 1892. He entered the college and received the A.B. degree in 1896.

An intense interest in the microscope led Dr. McClung into zoology and after graduation he become Dr. S. W. Williston's assistant in histology. He continued graduate work and received the A.M. degree in 1898. The first half of 1897–98 was spent in the laboratory of E. B. Wilson at Columbia University, and the following summer he worked with W. M. Wheeler at The University of Chicago. In 1902 he received the Ph.D. degree at Kansas, his famous paper, "The Accessory Chromosome: Sex Determinant?," was his thesis. At this time he was made Chairman of the newly formed Department of Zoology, a subject which was previously merely a satellite of the museum. From 1902 to 1906 he also served as acting Dean of the School of Medicine.

In 1912 he was appointed Professor of Zoology and Director of the Zoological Laboratory at the University of Pennsylvania. He became Professor Emeritus and was awarded an honorary degree of Doctor of Science in 1940. The following year he served as acting Chairman of the Department of Zoology at the University of Illinois. In 1942, Franklin and Marshal College gave him the honorary degree of Doctor of Science and in 1943 he became acting Chairman of the Department of Zoology at Swarthmore College.

Dr. McClung's early publications began at the age of 20 and dealt with pharmacy, microscopic technique and paleontology. It was through Dr. W. M. Wheeler that he became interested in Orthopteran cytology, a field of research in which he and his students made many important contributions to an understanding of the relation of chromosomes to heredity. His idea that sex is chromosomally determined seems to have been the first acceptable indication of a real correlation between chromosome constitution and body organization and it received world-wide attention.

In addition to his chromosome studies his writings reveal a wide range of interests beginning with pharmacy and including paleontology, microtechnique, photography and a variety of educational and philosophical subjects. With a group of collaborators he produced the ''Handbook of Microscopical Technique'' in 1929, followed by a second enlarged edition in 1937.

Dr. McClung was well known as a gifted and stimulating teacher. To him the mental development of the student was of far greater importance than the acquisition of specific knowledge. He displayed infinite patience, kindliness and human sympathy toward his students. He had a rare ability to analyze a student's mind and in private conferences he indicated to the student his special deficiencies and the methods of correction.

But Dr. McClung was far more than an investigator and teacher; he was versatile in interests and abilities. He contributed much to the improvement of the microscope and designed several features of Spencer's ''McClung Model.'' As an administrator he was constantly in demand. At Pennsylvania he was chairman of many committees, promoted curricular changes and the formation of the Faculty Research Committee. He was a member of the advisory boards of The Wistar Institute, the Morris Arboretum and the Cancer Research Institute. At Washington he was the first Chairman of the Division of Biology and Agriculture which organized the Union of American Biological Societies, and launched Biological Abstracts with Dr. McClung as President of the Board of Trustees from 1925 to 1933. He has always been editor of its section on Animal Cytology. Dr. McClung was devoted to the Marine Biological Laboratory. Since 1913 he was continuously a member of its Board of Trustees, a member of numerous committees and took an active part in securing funds for the new laboratory and the endowment.

Dr. McClung was an officer in many other organizations: he was President of the Central Branch of the American Society of Zoologists, 1910; the Combined Zoological Society, 1914; the National Society of Sigma Xi, 1917–21; the American Society of Naturalists, 1927; Beta Beta Beta Honorary Biological Fraternity, 1936 until his death; and Vice-President of Section F of the American Association for the Advancement of Science, 1926. He was also a member of the American Association of Anatomists, the Philadelphia Academy of Sciences, the American Philosophical Society, the National Academy of Sciences, and a foreign member of the Biological Society of Montevedeo.

From 1920 until his death he was managing editor of the Journal of Morphology.

Dr. McClung traveled considerably. In the interest of the Biological Abstracts he visited Europe in 1924, and had a sabbatical year (1927–28) at various laboratories there. He was delegate to the International Congress of Biology at Montevedeo, Uruguay (1930), and visiting Professor at Keio University, Tokio (1933–34). His last journey (1939) took him to South Africa and around the world to obtain Orthopteran material.

C. L. PARMENTER

THOMAS HUNT MORGAN

T. H. Morgan (Sept. 25, 1866–Dec. 4, 1945) was one of the leaders in the movement that has resulted in the predominantly physiological approach to biological problems. He was Professor of Experimental Zoology at Columbia for 24 years; and there have been few men more consistently devoted to the use of experimental methods.

Morgan is best known for his work in genetics; but zoologists recognize that his place in the history of the subject would be a high one even if he had never done genetical work. He was one of the early students of experimental embryology, where his work on artificial asters and on centrifuged eggs is particularly outstanding. The subject of regeneration will always be associated with his name. In both of these fields, and also in most of his writings on evolution, it appears that he was largely motivated by a desire to combat teleological interpretations and to replace them by mechanistic ones.

The range of subjects in which he made real contributions is a wide one; it is also interesting to note that he worked with many different kinds of animals — protozoans, coelenterates, ctenophores, flatworms, molluscs, marine and terrestrial annelids, crustaceans, pycnogonids, Drosophila, phylloxerans and aphids, sea-urchins, starfish, Balanoglossus, ascidians, Amphioxus, teleosts, amphibians, fowls, pigeons, rats, and mice. All these he kept in the laboratory, for he was remarkably skillful at keeping animals alive and in good condition. He was also a good naturalist; he knew where to collect animals, and could name a surprising variety of them at sight.

Morgan ordinarily used simple and direct methods of study. He was not inclined to use elaborate equipment — the early Drosophila work was done with hand-lenses rather than binocular microscopes, and the cultures were made up in a miscellaneous assortment of milk-bottles of various sizes, shapes, and provenience. He understood the necessity for expensive apparatus in much of modern experimental biology — but it was always an effort for him to support appeals for such apparatus.

For many years he spent his summers at Woods Hole, where he was one of the group of leaders that had so much to do with the development of American biology. The influence of this laboratory is without parallel in other sciences, and in other countries has been approached only by that of Naples; no small part of that influence was due to Morgan, who, like several others of the Woods Hole group, was himself strongly influenced by early stays at Naples.

Morgan received his B.S. from the University of Kentucky in 1886; he was attracted to The Johns Hopkins University, then emerging as a new type of graduate institution under the leadership of President Gilman, and received his Ph.D. there in 1890. From 1891 to 1904 he was Professor of Biology at Bryn Mawr, from 1904 to 1928 Professor of Experimental Zoology at Columbia. In 1928 he organized a new department at the California Institute of Technology, where he became Professor of Biology. His work with Drosophila began in 1909; since 1915 it has been supported in part by a grant from the Carnegie Institution of Washington. This grant was used by Morgan to support a group of assistants — at first C. B. Bridges and the writer, later J. Schultz. His policy here was characteristic: he believed in doing his own work, and never entrusted critical parts of it to technical assistants. When funds were available to support work in a field, his method was to look for competent men and then put them on their own responsibility, rather than to get a larger group working under more direct supervision.

A. H. STURTEVANT

EDWIN EUSTACE REINKE

Dr. Edwin E. Reinke, Professor of Biology and Chairman of the Division of Natural Sciences in Vanderbilt University, died of an acute illness at his home in Nashville, Tennessee, on January 25, 1945, in his fifty-seventh year. Professor Reinke was well-known in scientific circles throughout the southeast, and it was in large part through his efforts that Vanderbilt University has attained a prominent position in southern pre-medical education in step with the development of the Vanderbilt University School of Medicine.

Born of American parentage in Jamaica, B. W. I., Dr. Reinke was educated in the Moravian Preparatory School at Bethlehem, Pennsylvania, and in undergraduate collegiate studies at Lehigh University. Earning the degree of Ph.D. for investigations in cytology at Princeton University under the direction of Professor Conklin in 1913, he then became an Instructor in Biology at Rice Institute, Houston, Texas, at the time when Professor Julian Huxley headed the department in that new institution. During the period 1912–1915, Dr. Reinke was a Research Associate of the Carnegie Institution of Washington at its famous Tortugas Laboratory, and was in close association with Dr. Alfred G. Mayer. His studies of the unusual phenomena associated with spermiogenesis in the mollusca resulted in several splendid papers which demonstrated a great capacity for detailed work explained with perception and clarity.

On moving to Tenessee in 1915, he turned his attention to the development of pre-medical education and placed the Department of Biology at Vanderbilt University on a firm foundation. In 1928 Dr. Reinke and his colleagues moved into Buttrick Hall, a new building for biology which incorporated many of his own ideas on construction and arrangement. Close attention to the demands of effective teaching left little time for continuing research in line with the earlier days, but association with students and colleagues gave rise to a furtherance of investigations in these same fields. He never lost interest in the advancement of cytology, and inspired many a student with his elucidation of zoology and evolution in their relations to pure and applied biology. Maintaining a summer home in Highlands, North Carolina, he was Director of the Highlands Museum and Biological Laboratory from 1929 to 1935. Long a member of the American Society of Zoologists, he maintained constant interest in that organization. In 1938–1939 he was President of the Association of Southeastern Biologists. His investigations comprised the studies of spermic dimorphism in the prosobranch snails, taxonomy of the salamanders, and investigations in the causation of ''water-drive'' during the life-cycle of Triturus.

C. S. SHOUP

BERTRAM GARNER SMITH

Bertram Garner Smith, Professor of Anatomy in New York University College of Medicine until his retirement in 1942, died at his home in Albuquerque, New Mèxico, on July 30, 1945.

Born in Painesville, Ohio, on October 7, 1876, Dr. Smith received his early education in the public schools and in Youngsville High School. After graduating from Pennsylvania State Normal School in 1896, he began his teaching career in the Warren, Pennsylvania, high school, and continued his training in the summer sessions at Cornell University. In 1904, as Assistant in Zoology, he entered the University of Michigan, where he received the A.B. degree in 1907. He served subsequently as Instructor in Lake Forest College (1907), Syracuse University (1907–09) and the University of Wisconsin (1909–11). Obtaining the Ph.D. degree from Columbia University in 1912, Dr. Smith became Assistant Professor and later Associate Professor in Michigan State Normal College, where he remained until he came to New York University as Associate Professor of Anatomy in 1921. After his promotion in 1929, he served until his retirement as Professor of Anatomy in charge of the courses in histology and embryology.

Dr. Smith's research interests ranged widely through anatomy, embryology and cytology, and his numerous papers spanned the years from 1905 to his retirement. An important editorial contribution was his service for 10 years as one of the chief editors of the Bashford Dean Memorial series on the anatomy and natural history of the frilled shark, published by the American Museum of Natural History.

Although rather retiring, almost to the point of shyness with casual acquaintances, Dr. Smith is remembered by his students and colleagues for a friendly and gracious personality which leavened his teaching and enriched his contacts with his associates.

R. P. HALL

FRANCIS BERTODY SUMNER

On September 6, 1945 the American Society of Zoologists lost through death one of its outstanding senior members, Francis Bertody Sumner, Professor of Biology (Emeritus) at Scripps Institution of Oceanography, University of California. In 1938, as Vice-President of the American Association for the Advancement of Science, he served as the society's chief. The esteem which American zoologists expressed in electing Dr. Sumner to this post confirmed the recognition which was conferred on him through appointment to fellowship in the American Association, the California Academy of Sciences and the San Diego Society of Natural History and, more particularly, through election to membership in the Philadelphia Academy of Natural Sciences, the American Philosophical Society and the National Academy of Sciences.

Sumner was born in Connecticut on August 1, 1874, was reared in California and Colorado and was college-trained at Minnesota and Columbia. Henry F. Nachtrieb crystallized his strong inclinations toward natural history into determination to become a biologist. Great leaders at Columbia — Edmund B. Wilson, Henry Fairfield Osborn, Bashford Dean and James McKeen Cattell — furthered his training and fixed his inspiration.

Early mastery of observation, analysis and experimentation was exhibited in Sumner's doctoral thesis, on the relation of Kupffer's vesicle to gastrulation and concrescence. Rigorous intellectual honesty, free-thinking, thoroughness and precision were evidenced throughout his long ever-sustained research career. His contributions were marked by variety and originality and all, perhaps with the exception of his early attempt to test the inheritance of acquired characters, by definiteness and significance. He supervised model biotic surveys of the Woods Hole region and of San Francisco Bay. To him belongs a very large share, in most items nearly all, of the credit for the determination of the background-simulating capacities of flatfishes, the ability of Fundulus to adjust itself to changes in salinity, the acclimatization of fishes to low and high temperatures, the genetic fixity of adaptive characters in mice of the genus Peromyscus, the occurrence of simple mutations but the complexity of the inheritance of specific and racial characters in wild mammals, the survival value of individual color adaptations, and, as his final research, the quantitative changes in pigments of fishes under different optical stimuli: a distinguished record indeed.

Sumner the man — nature lover, sham hater, somewhat reticent, always considerate, avoider of conventions, ever a courageous crusader with deep social consciousness — has been ably and frankly, though in some respects too modestly, analyzed in "The Life History of an American Naturalist," a thought-provoking autobiography that appeared just in time to be appreciated by the author. Colleagues sought his frank criticism and intimate friends cherished his witty conversation and valued advice. His personal influence as well as his contributions to science live on.

CARL L. HUBBS

HERBERT EUGENE WALTER

Herbert Eugene Walter, Professor of Biology Emeritus at Brown University, died October 1, 1945 at the age of 78. He came to Brown in 1906 and retired from active teaching in 1937.

He was born in Burke, Vermont, April 19, 1867, graduated from Bates College in 1892, received the A.M. degree at Brown in 1893, studied in Freiburg, Germany, in 1893–94 and in 1903, and received his Ph.D. degree at Harvard University in 1906. He was awarded the honorary degree of Sc.D. by Middlebury College in 1934 and by Bates College in 1937.

He was primarily a teacher. At Brown, he taught comparative anatomy and genetics to large classes of enthusiastic students. For several years before coming to Brown, he had taught biology in a Chicago high school, and from 1906 to 1927 he conducted a famous course in field zoology in the Biological Laboratory at Cold Spring Harbor. From 1917 to 1927 he was Assistant Director of that laboratory. In his many years of teaching, he never lost his enthusiasm for his subject nor for his students. Teaching was with him a very personal matter. His students remember him not only for his vivacious teaching but for his warm personal friendship. Yet his exposition was conspicuously systematic and thorough.

He was the author of several books, all of which grew out of his teaching: Wild Birds in City Parks in collaboration with his wife, Alice Hall Walter; Genetics (1913); The Human Skeleton (1918); and The Biology of the Vertebrates (1927).

Into his textbooks he put his own personality and native wit, which made them attractive reading, in addition to being a sound introduction to serious subject matter.

The outstanding features of his personality were those characteristics of the people of northern Vermont where he and generations of his forebears had lived. He had a keen insight into human nature. Charitable with the faults and shortcomings of others, he had a knack of bringing out the best in those who worked with him. Reluctant to criticize their work, he was quick to recognize and encourage merit and achievement. To all who came close to him as student or as colleague, it was delightful and profitable to have known him.

Through his textbooks and through his teaching at Brown and at Cold Spring Harbor, whence his students have carried his methods far and wide, he has exerted a profound and important influence on the teaching of biology in America.

J. WALTER WILSON

HENRY BALDWIN WARD

Henry Baldwin Ward ended his distinguished and extremely active career on November 30, 1945. The preceding day he had spent in customary pursuits and without forewarning a heart attack carried his slumber into death. To his extremely broad circle of colleagues and acquaintances he will long be remembered for the diversity of his interests. To many he was known as a teacher, an organizer, a writer, an administrator, or an investigator; but some of his intimate acquaintances remember him best as a lover of the out of doors where hiking, soft ball, heated discussions and group singing around the campfire brought to a close the almost invariable Saturday afternoon of recreation.

Henry Baldwin Ward, son of Richard Halsted and Charlotte Allen Baldwin Ward, was born in Troy, New York, on March 4, 1865. Upon graduation from Williams College (A.B., 1885) he served for 2 years as a science teacher in the Troy High School before entering upon graduate study. The years 1888 to 1890 he spent in Europe where he formed many personal and professional contacts which he cherished throughout life. He studied in the universities of Göttingen, Freiburg, and Leipzig and in the biological stations of Naples, Heligoland, and Ville-Franche-sur-Mer. On return to America he continued his graduate studies at Harvard.

In his writing as well as in his lectures he often referred to Joseph Leidy as the founder of parasitology in America but measured in terms of workers in the field of parasitology who were trained in his laboratories there are few who would fail to accord to Professor Ward the distinction of having impressed his influence upon teaching and research in parasitology in America more than any other individual. Trained in the world-renowned laboratory of Rudolf Leuckart in Leipzig, Professor Ward established in the Universities of Nebraska (1893–1909) and Illinois (1909–1933) centers for the study of parasites which have profoundly influenced every modern trend in the fields of parasitology and tropical medicine.

A casual listing of his diverse interests and activities would fail to reveal that in his life events and interests usually had definite causal sequences and climaxes, of which a few instances will be mentioned. Microscopy and invertebrate zoology were lifelong interests traceable to the influence of his father and to his training under Professor E. L. Mark at Harvard (A.M. and Ph.D., 1892). His early teaching and research interest in fresh water biology and fisheries found initial active expressions while he was on the faculty of the University of Michigan (1892–1893) and in the employ of the Michigan Fish Commission (summer of 1894). These activities remained with him to share his enthusiasm for the field of parasitology. The fruition of Professor Ward's attention to microscopy and limnology was the monumental volume on Freshwater Biology (Wiley, 1918). The climax of his early interest in fishery biology was his long term program of studies on the Alaska salmon, chiefly in cooperation with the U. S. Bureau of Fisheries.

Beginning in the late years of his active association with the University of Illinois and continuing with increasing drive after he became Professor Emeritus (1933) his attention became directed more and more to the problems of education in conservation of nature and wildlife.

In every task which he assayed, he became so deeply engrossed that he attained for himself recognition for active leadership. He gave time and effort generously to the societies and organizations with which he became affiliated.

He was President of the Central Branch of the American Society of Zoologists (1912–1913). Small as well as large and important scientific groups shared his attention. Throughout his professional life he was an ardent supporter of the American Microscopical Society (Secretary-Editor 1898–1904, President 1905) whose early meetings he attended with his father in the days when the microscope was as much an object of curiosity as a tool for research. He was national President of Sigma Xi (1922–24) after serving as general Secretary (1895–1922) during the rapid rise of that society to prominence and influence. He compiled the Quarter Century Record and History of the society (1913) and for many years was a member of the editorial committee of the Sigma Xi Quarterly. He served the American Association for the Advancement of Science in numerous capacities and became its Permanent Secretary (1933–1937). He championed the cause of conservation as President of the Izaak Walton League of America (1928–1930).

He became Dean of the University of Nebraska College of Medicine (1902–1909) and thereafter he retained a very active interest in medical education. Medical zoology, parasitology and tropical medicine were all intimately intertwined in his thinking.

Largely as a personal venture he founded the Journal of Parasitology (1914) which exercised an important role in unifying the interests of parasitologists on this continent. His leadership in this field was acknowledged when he was made the first President of the American Society of Parasitologists (1925), the organization which ultimately took over the Journal. With his colleagues S. A. Forbes and T. J. Burrill he was influential in having the University of Illinois establish the Illinois Biological Monographs for which the three served as an editorial committee.

His honors and memberships include many of the honorary and professional societies in foreign countries. An enumeration of all of the editorial assignments and offices in professional organizations held by Professor Ward would extend this memoir far beyond the available space. The few that have been selected for listing speak eloquently of the diversity of his interests.

The University of Cincinnati honored him with the degree of Sc.D. in 1920 and the following year Williams College, his Alma Mater, bestowed the same degree upon him. The Universities of Oregon (1932) and Nebraska (1935) both honored him with the degree of LL.D.

With apparent intention he chose sound teaching, broad professional contacts and generous service activities to stand among his chief contributions to American science.

In his immediate family, Professor Ward is survived by his wife, Harriet Blair Ward, and their two daughters, Mrs. Cecilia Tanton and Charlotte Baldwin Ward.

HARLEY J. VAN CLEAVE

The foregoing resolutions were adopted by a rising vote and the Secretary was instructed to send copies to the nearest relatives and to publish the resolutions in the Proceedings of the Society.

3. *Report of the Treasurer.* The Treasurer presented the following report for the year 1945:

Credits:			
Cash balance from 1944	$ 882.98		
U. S. $1,000 Savings Bonds M305107D and M305109D; present redemption value	1,740.00		
Receipts from dues during 1945	3,722.05		
Total			$6,345.03
Expenditures:			
Secretary's expenses:			
Secretarial services	$ 53.03		
Stenographic supplies	24.75		
Postage and express	28.65		
Telephone and telegraph	12.80		
Assistance	205.40		
		$324.63	
Treasurer's expenses:			
Secretarial	$ 112.50		
Printing	64.45		
Postage	43.50		
		220.45	

The Wistar Institute:			
Reprints Memorial Resolutions	$ 13.50		
Reprints Programs and Abstracts	1,132.30		
Postage	59.99		
		1,205.79	
Bank charges:			
Iowa State Bank	$ .94		
First National Bank, Bloomington, Ind.	.83		
		1.77	
Union of American Biological Societies for expenses as noted at Cleveland		15.00	
		$1,767.64	
Balance, December 31, 1945:			
Checking account	$2,837.39		
Two U. S. Savings Bonds at $870.00	1,740.00		
Total		4,577.39	6,345.03

Gross receipts from dues, increase over 1944	$ 636.25
Debits on 1945 paid in 1946	1,963.63
Decrease in assets in 1945	1,327.38
Unattached assets on December 31, 1945	2,613.76

4. Report of the Auditing Committee. The Auditing Committee consisting of Fernandus Payne and T. M. Sonneborn presented the following report:

We have examined the books and accounts of the Treasurer of the American Society of Zoologists and find them to be in order and correctly balanced. The Treasurer's annual report is in accord with the books up to December 31, 1945.

Signed: FERNANDUS PAYNE
T. M. SONNEBORN

It was voted to accept and approve the reports of the Treasurer and of the Auditing Committee.

5. Report of the Executive Committee.

(a) Support of Biological Abstracts. The Committee recommended support of Biological Abstracts for 1946 on the same plan as for 1945, namely, that $2.00 from each membership fee of $4.00 be paid to Biological Abstracts.

It was voted to approve this recommendation.

(b) Appropriation for Biologists' Smoker. The Committee recommended that $15.00 be appropriated, as in past years, toward defraying the expenses of the Biologists' Smoker.

It was voted to approve this recommendation.

(c) Tenure of Society Representatives on the Council of the American Association for the Advancement of Science. In order to serve more effectively on the Council of the American Association for the Advancement of Science, the Committee recommended that the tenure of Society Representatives be increased from 1 to 2 years, one representative to be elected each year.

It was voted to approve this recommendation.

(d) Remission of dues. A resolution suggesting that payment of dues by members in the armed services and foreign members be suspended for the duration was presented and passed early in 1943.

Since the emergency has now passed for most of those concerned, the committee recommended that this resolution be revoked at the end of 1945 for all members who were exempt from payment of dues, excepting foreign members in the devastated countries of Europe and Asia.

It was voted to approve this recommendation.

e) Election of New Members. In accordance with the stipulations in the Constitution regarding membership, the Committee recommended the following candidates for election to membership in the Society.

ACTIVE MEMBERS

(Formerly Associate Members)

Allen, Thomas Hunter
Balamuth, William
Bookhout, Cazlyn Green
Bullock, Theodore Holmes
Deane, Helen Wendler
Emerson, Henry Seelye
Evans, Francis Gaynor
Farner, Donald S.
Fuller, John Langworthy
Gregg, Robert Edmond
Guhl, Alphaeus Matthew
Hauschka, Theodore Spaeth
Hulpieu, Harold R.
Kent, George C., Jr.
Novikoff, Alex Benjamin
Rose, S(ylvan) Meryl
Russell, William Lawson
Scharrer, Berta
Schmieder, Rudolf G(ustav)
Scott, John Paul
Shoemaker, Hurst Hugh
Strohecker, Henry Frederick
Tittler, Irving Albert
Watterson, Ray Leighton
Wilber, Charles Grady

(Not Formerly Associate Members)

Aronson, Lester Ralph
Beaver, Paul Chester
Beutner, Reinhord H.
Carson, Hampton Lawrence
Chandler, David C.
Charles, Donald R.
Chase, Herman Burleigh
De Bruyn, Peter P. H.
Fisher, Kenneth Clarke
Harris, Morgan
Harrison, James A(lexander)
Ives, Philip Truman
Kollros, Jerry John
Linsdale, Jean Myron
Metz, Charles Baker
Nalbandov, Andrew Valdimir
Phelps, Austin
Power, Maxwell Elliott
Reid, Malcolm W(illard)
Stone, Wilson Stuart

NEW ASSOCIATE MEMBERS

Baker, E(dgar) G(ates) Stanley
Berry, Levette Joe
Blake, Irving H(ill)
Blivaiss, Ben Burton
Bonnet, David Dudley
Braddock, James Conger
Busch, Karl H. D.
Cieslak, Edwin S.
Clark, Hugh
Cole, LaMont C.
Collias, Nicholas Elias
Crouse, Helen V(irginia)
Dougherty, Ellsworth Charles
Ferguson, Frederick Palmer
George, Ira D.
Greiff, Donald
Hardin, Garrett J(ames)
Hudson, George Elford
Lansing, Albert I.
Lewis, Lillian L. Burwell
LeFevre, Paul Green
Littleford, Robert Anthony
Maldonado-Koerdell, Manuel
Merwin, Ruth M.
Michalski, Joseph Vitold
Moog, Florence
Pitelka, Frank Alois
Ring, John Robert
Rodgers, Thomas Lathan
Rostorfer, Howard Hayes
Rothchild, Irving
Russell, Elizabeth Buckley Shull
Sheehan, John Francis
Test, Frederick H(arold)
Wheeler, Norman C.
Wolfson, Albert

It was voted that all candidates be elected as recommended by the Executive Committee, and the Secretary was instructed to cast one ballot in their favor.

6. Changes in membership. For the information of the Society the Secretary reported the following changes in membership which occurred during the past year. The following summary includes the above newly elected candidates:

Deaths	12	New associate members	36
Resignations	15	Reinstated	0
Dropped	36	New active members	20
	—		—
	63		56

Total enrollment in last report	1,087
Loss in enrollment	7
Total enrollment at present	1,080

7. Election of officers. The Nominating Committee consisting of M. H. Jacobs, Chairman; A. E. Emerson, and J. S. Nicholas, presented the following report:

President, 1 year: D. E. Minnich.
Vice-President, 1 year: E. G. Butler.
Secretary, 3 years: L. V. Domm.
Executive Committeeman, 5 years: A. S. Pearse.
Society Representatives on the Council of the American Association for the Advancement of Science, 2 years: A. Elizabeth Adams; Waldo L. Schmitt.
Society Representatives on the Council of the Union of American Biological Societies, 1 year: H. B. Adelmann; H. H. Plough.
Society Representatives on the Advisory Editorial Board of Biological Abstracts: M. Demerec; R. K. Enders; R. G. Kempton, E. J. Lund.
Society Representative on the Executive Committee of the Biological Stain Commission: S. I. Kornhauser.
Managing Editor of the Journal of Morphology, 5 years; E. G. Butler.
Members of the Editorial Board of the Journal of Morphology, 3 years: G. Fankhauser; T. M. Sonneborn; D. M. Whitaker.

It was voted that the above named candidates for office be elected and the Secretary was instructed to cast one ballot in their favor.

8. *Committee Reports.* (a) The War Emergency Subcommittee of the Executive Committee consisting of C. G. Hartman, Chairman; M. H. Jacobs, J. S. Nicholas, L. L. Woodruff, T. S. Painter, Sewall Wright, and A. S. Pearse and L. V. Domm, Ex officio, presented the following final report:

The efforts of the War Emergency Committee, in the early years of the war, were centered on efforts to make the Selective Service truly selective, to the end that trained men might more generally be allocated to activities in line with their training: entomologists for insect control, bacteriologists and parasitologists for hospital work, nutritionists for food problems, botanists for rubber, wood supplies, etc. Gradually the Army and Navy (the Army very slowly) learned what Russia, Germany and Britain knew and practiced from the beginning of this war — that, while you might make a truck driver out of a scientist in a week, it takes years to develop a scientist, even a biologist!

It would be interesting to know how many biologists were salvaged for technical work through the efforts of heads of departments, perhaps aided by the agitation of our committee. One wonders, too, how many scientifically trained men were recalled out of the rank and file for expert work which only they were trained over the years to do.

It is a question whether the men who determine the policies of the armed forces have become fully conscious of the value of the biologist to preparedness. It is difficult to have confidence in the social consciousness and scientific insight of the military in view of its past stupidities and exhibitions of shortsightedness. With the passing of this committee, the parting suggestion of your chairman is this, that the role of the biologist in national preparedness, for any emergency, be made the subject of thorough study by the projected Confederation of Biological Societies of America, launched March 29, 1946, at Saint Louis.

(b) The following report by S. I. Kornhauser, Representative of the Society on the Executive Committee of the Biological Stain Commission, was presented:

> As the representative of the American Society of Zoologists on the Biological Stain Commission, I wish to make the following report for the year 1945.
>
> The Commission having become a corporation with assets of about $160,000 made the work of the treasurer, your representative, more arduous along business rather than scientific lines.
>
> We had a meeting of the Executive Committee and the Commission in Philadelphia and New York in February. The main subject of discussion concerned the possibility of starting original investigation on stains and their application supported by the Commission. The idea of the Committee on Research was that fundamental chemical and physical knowledge of biological stains was necessary to make staining a science rather than an art, and that such a program should be initiated as soon as conditions warranted. The return from Army service of Dr. H. A. Davenport, formerly the Anatomists' representative on the Commission has permitted us to begin this work in a modest manner. Dr. Davenport has received a grant from the Commission to study the fixation of tissues for neurological staining.
>
> In June, the Executive Committee met in Geneva, New York, to survey the situation there relative to continuing our headquarters at Geneva where much of the testing of dyes and the publication of the Commission literature is done. Later in the summer we were able to conclude a contract with Cornell University which permitted the continuation of the work under favorable conditions at Geneva.
>
> The publications of the Commission have survived the war successfully and during the year 1946 the Fifth Edition of Biological Stains should appear. This book together with Stain Technology and the other publications printed at Geneva are now the property of the Biological Stain Commission.
>
> I should again like to invite the members of the American Society of Zoologists to send their papers on stains and microscopical technique to Stain Technology for publication and also invite your criticism of the work of the Commission, and the performance of certified dyes. I should welcome your suggestions of problems which should be attacked by us.

(c) The Motion Picture Committee, consisting of Robert Chambers, Jr., Chairman; Ralph Buchsbaum, and O. W. Richards, working with E. J. Farris, representative of The Wistar Institute of Anatomy and Biology, submitted the following report:

Because of the pressing problems caused by the war, the Motion Picture Committee has not reviewed any films since the last meeting.

The following suggestions are presented for consideration and approval by the Society:

1. A Committee of three individuals should be appointed annually to work with The Wistar Institute in evaluating and reviewing films in Biology.
2. The Committee should maintain information relative to motion picture films in biology that may be of value to individuals interested in using them.
3. The Committee should solicit films from producers and distributors for review purposes, the reviews to be published by The Wistar Institute.
4. The members of the American Society of Zoologists are requested to submit titles and any new films to the Committee for review.
5. Films of outstanding value should be recommended by the Committee to The Wistar Institute for distribution, thus making them available to all members of the Society.

It was voted that the foregoing committee reports be approved.

(d) National Research Foundation Act of 1945. For information the Secretary reports the following: In September 1945 Dr. A. S. Pearse appointed a committee to prepare a statement regarding the position of zoology in the proposed legislation pertaining to a National Science Program. The committee prepared the following statements, copies of which were sent to Senators Harley M. Kilgore and Warren G. Magnuson and to the committee representing the biological sciences at the hearings:

1. Zoology, which is a basic science, has as its aim the understanding and the increase of knowledge of all animal life, including the physical nature of man. It is a foundation science, the root from which have sprung such applied sciences as animal breeding, economic entomology, fish and wild life management. Moreover, it has contributed substantially to the progress of medical science, psychology, and sociology. To cite but one example, our struggle against the parasitic diseases during World War II would have been ineffectual without the knowledge of structure, physiology, and life histories of parasites gained through pure zoological research.
2. The science of zoology pioneers in investigations dealing with animals, both as to their nature and environmental relationships, regardless of whether such knowledge serves any utilitarian purpose. In the past the United States Government has encouraged and supported those branches of zoology which have an economic or applied bearing, as witness the U. S. Bureau of Entomology, the Bureau of Animal Industry, the Fish and Wild Life Service, and similar agencies, but has done little or nothing in supporting zoological research which is basic for the advances made in the applied field.

3. The natural sciences are so closely inter-related that advances in one often depend upon advances in the other. Zoological discoveries are made possible through techniques developed by the physical sciences, and to a certain degree the advances in the physical sciences are stimulated by new discoveries in zoology. For instance, new discoveries in zoology have given the chemist clues to previously unknown chemical substances such as the hormones. Furthermore, many of the most important problems confronting science today are borderline problems which demand the cooperation of scientists from different fields, and therefore the scientific front must be advanced all along the line.

4. For the reasons presented above, we strongly recommend that zoology be not regarded merely as a subsidiary of medicine or some other applied science, but that it be given equal rank with the other basic natural sciences in the National Program of Scientific Research.

5. In making available and distributing information on all discoveries produced in the course of federally financed research it is recommended that (1) existing agencies be used and supported — as, for instance, the international abstracting service of Biological Abstracts; (2) existing research journals be expanded; and (3) financial support for publication of original researches be provided.

6. We approve the proposal to grant aid for basic research to non-profit research and educational institutions, as it is particularly this class of institution that has in the past contributed most to basic research.

7. We highly approve the proposal to strengthen basic research through the provision of scholarships and fellowships to competent young men and women in higher education.

8. We strongly urge that direction and administration of the proposed *National Research Foundation* be throughout the responsibility of trained scientists. Only those trained in the subject can fully appreciate the relative importance of specific needs of research in animal biology, and of the quality of the research completed.

October 9, 1945 Signed: BENTLEY GLASS
EDWARD G. REINHARD
B. H. WILLIER, Chairman

9. Proposed Federation of Biological Societies. The Union of American Biological Societies in meeting at St. Louis requested the adoption of a resolution which was presented to the meeting as follows:

Resolved, that the American Society of Zoologists endorse the formation of an organization of biologists on the basis of the following general propositions:

1. To provide a means for executing our public responsibilities as biologists and scientists, so that the freedom essential to the progress of science and public welfare be insured.

2. To safeguard the professional interests of biologists and to assist in providing the material means for the promotion of biological research as well as to provide such services as may be necessary to expedite this program.

In order to facilitate the formation of such an organization it was further resolved that:

1. The President be instructed to appoint a committee to cooperate in any way possible with other organizations working toward this goal.
2. The Executive Committee be authorized to appropriate funds, if needed, not to exceed $100.00 to aid in the formation of this organization.

The resolutions were approved by unanimous vote.

10. MacMahon Bill — S. 1717. The following telegram was submitted with the request that it be approved and sent to Senator MacMahon:

> We heartily endorse the MacMahon bill S. 1717, without any amendment decreasing civilian control of atomic energy, and register our opposition to the May-Johnson bill and any other legislation embodying military control of atomic energy.

The above telegram was approved and the Secretary instructed to send it to Senator MacMahon.

11. Status of trained personnel in animal sciences. The following is a resume of a letter dated March 12, 1946 from Dr. Robert F. Griggs, Chairman, Division of Biology and Agriculture, National Research Council, to Dr. A. S. Pearse, President, American Society of Zoologists:

> Assuming that there is a shortage of trained personnel in the animal sciences which will last for some years, it is suggested that a committee be appointed to make a careful survey and assessment of conditions in zoology. It is felt by representatives of some of the sciences that a concerted study of supply and demand, followed by efforts to meet the demand, ought to be undertaken in each of the sciences. A study of this sort was recently made in agricultural sciences and similar studies are contemplated in the various fields of engineering. When these reports become available, persons in charge of assisting in the training of technical personnel might readily conclude that the needs were greater in engineering than in the other sciences. Dr. Griggs says, ''I am thinking particularly that if and when a fellowship and scholarship program is set up by the government under a National Science Foundation, or otherwise, those responsible for the administration of such a program would naturally look to existing data and their decisions as to allocation of their funds would be influenced largely by available data.''
>
> The National Research Council offered to assist such a committee in various ways especially by holding occasional meetings at which results could be presented and problems discussed.

The Society voted approval of the proposed personnel study and recommended that the Division of Biology and Agriculture, National Research Council make such a study in the animal sciences.

12. Nominating Committee for 1946. The President appointed the following nominating committee to prepare a slate of officers for the coming year: W. H. Cole, Chairman; H. W. Manter, and Charles Packard.

13. Vote of Appreciation. It was voted to express the sincere thanks of the Society to the members of the local committee, consisting of Viktor Hamburger, Chairman; Father Paul W. Carroll, H. L. Carson, D. Greiff, Alphonse Schwitalla, H. B. Steinbach, and H. Stalker, and to Saint Louis University and Washington University for the arrangements and cordial hospitality which served under abnormal conditions to make the Saint Louis meeting a success.

The meeting was adjourned.

L. V. Domm,
Secretary.

LIFE AND ACTIVE MEMBERS

(* indicates life members of the Society; † indicates charter members of the American Morphological Society; mail addresses are in italics.)

Ackert, James Edward, A.B., A.M., Ph.D. (Illinois), Professor and Head of the Department of Zoology, and Parasitologist, Agricultural Experiment Station; Dean, Graduate School; *Kansas State College, Manhattan, Kan.*

Adams, A. Elizabeth, B.A. (Mount Holyoke), M.A. (Columbia), Ph.D. (Yale), Professor of Zoology, *Mount Holyoke College, South Hadley, Mass.*

Adamstone, Frank Bolton, B.A., M.A., Ph.D. (Toronto), Professor of Zoology, *345 Natural History Building, University of Illinois, Urbana, Ill.*

Adelmann, Howard Bernhardt, A.B., A.M., Ph.D. (Cornell), Professor of Histology and Embryology, Chairman, *Department of Zoology, Cornell University, Ithaca, N. Y.*

Adolph, Edward Frederick, A.B., Ph.D. (Harvard), Associate Professor of Physiology, School of Medicine, *University of Rochester, Rochester, N. Y.*

Agersborg, H(elmer) P(areli von Wold) K(jerschow), B.S., M.S. (Washington), M.A. (Columbia), Ph.D. (Illinois), Professor of Biology and Chairman of the Department, *Department of Biology, McKendree College, Lebanon, Ill.*

Alexander, Albert, B.A. (St. Stephen's), M.A., Ph.D., M.D. (Harvard), Research Assistant, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Alexander, (Edward) Gordon, A.B. (Central), A.M., Ph.D. (Princeton), Professor of Biology, *University of Colorado, Boulder, Colo.*

Allee, Warder Clyde, S.B., LL.D. (Earlham), S.M., Ph.D. (Chicago), Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Allen, Bennet, Ph.B. (De Pauw), Ph.D. (Chicago), Professor of Zoology, University of California, Los Angeles, *909 Micheltorena St., Los Angeles, Calif.*

Allen, Thomas Hunter, B.A., Ph.D. (Iowa), Instructor in Physiology, *Department of Physiology, College of Physicians and Surgeons, Columbia University, 630 West 168th St., New York 32, N. Y.*

Allen, William Ray, A.B., A.M., Ph.D. (Indiana), Professor of Zoology, *Department of Zoology, University of Kentucky, Lexington, Ky.*

Altenburg, Edgar, A.M., Ph.D. (Columbia), Assistant Professor of Biology, *Rice Institute, Houston, Texas.*

Anderson, Bertil G., A.B. (Augustana), M.S., Ph.D. (Iowa), Associate Professor of Zoology, *Department of Botany and Zoology, West Virginia University, Morgantown, W. Va.*

Andrew, Warren, A.B. (Carleton), M.S. (Brown), Ph.D. (Yale, Illinois), M.D. (Baylor), Associate Professor of Histology and Embryology, *Southwestern Medical Foundation School of Medicine, Dallas, Texas.*

†*Andrews, Ethan Allen, Ph.B. (Yale), Ph.D. (Johns Hopkins), Professor of Zoology, Emeritus, *The Johns Hopkins University, Baltimore, Md.*

Andrews, Frederick Newcomb, B.S., M.S. (Massachusetts State), Ph.D. (Missouri), Associate in Agricultural Experiment Station and Assistant Professor of Animal Husbandry, Purdue University, *Agricultural Experiment Station, Purdue University, Lafayette, Ind.*

Angerer, Clifford A., A.B. (Columbia), Ph.D. (Pennsylvania), Assistant Professor of Physiology, *Department of Physiology, Ohio State University, Columbus, Ohio.*

Arey, Leslie Brainerd, Ph.D. (Harvard), Sc.D. (Colby), Robert L. Rea Professor of Anatomy and Chairman of the Division of Anatomy, *Northwestern University Medical School, 303 E. Chicago Ave., Chicago, Ill.*

Armstrong, Philip B., B.S. (Massachusetts State), M.D. (Cornell), Professor of Anatomy, *Department of Anatomy, College of Medicine, Syracuse University, Syracuse 10, N. Y.*

Aronson, Lester Ralph, B.A., M.A. (Cornell), Ph.D. (New York), Assistant Curator, *Department of Animal Behavior, American Museum of Natural History, Central Park West at 79th Street, New York 24, N. Y.*

Bagg, Halsey Joseph, B.S., A.M., Ph.D. (Columbia), *Bagg Research Laboratory, Yorktown Heights, N. Y.*

Baitsell, George Alfred, B.S. (Central College, Iowa), M.A., Ph.D. (Yale), Professor of Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

Bailey, Percy Laurance, Jr., A.B., Ph.D. (Brown), Assistant Professor of Biology, *College of the City of New York, Convent Ave. and 139 St., New York, N. Y.*

Baker, Horace Burrington, B.S., Ph.D. (Michigan), Professor of Zoology, University of Pennsylvania, *Zoological Laboratory, University of Pennsylvania, Philadelphia 4, Pa.*

Balamuth, William, B.S. (College of the City of New York), Ph.D. (California), Assistant Professor of Zoology, *Department of Zoology, Northwestern University, Evanston, Ill.*

Ball, Gordon Harold, B.S., M.S. (Pittsburgh), Ph.D. (California), Associate Professor of Zoology, *University of California, Los Angeles 24, Calif.*

Ball, Stanley Crittenden, Ph.B., Ph.D. (Yale), Associate Professor of Biology and Curator of the Zoological Collection, *Peabody Museum, Yale University, New Haven, Conn.*

Ballantine, Robert, A.B., Ph.D. (Princeton), National Research Fellow in Zoology, *Department of Zoology, Columbia University, New York 27, N. Y.*

Bangham, Ralph Vandervort, A.B. (Wilmington), B.S., A.M. (Haverford), Ph.D. (Ohio State), Professor of Biology and Head of the Department, *Wooster College, Wooster, Ohio.*

Barnes, Thomas Cunliffe, B.A. (Cornell), D.Sc. (Harvard), Associate Professor of Physiology, *Hahnemann Medical College, Philadelphia, Pa.*

Barrows, William Morton, B.S. (Michigan Agricultural College), S.B., S.M., S.D. (Harvard), *Department of Zoology and Entomology, Ohio State University, Columbus, Ohio.*

Bartelmez, George William, B.S. (New York University), Ph.D. (Chicago), Professor of Anatomy, *Hull Anatomical Laboratory, The University of Chicago, Chicago 37, Ill.*

Barth, Lester G., A.B. (Wayne University, Detroit), A.M. (Michigan), Ph.D. (Chicago), Associate Professor of Zoology, *Department of Zoology, Columbia University, New York, N. Y.*

Bartsch, Paul, B.S., M.S., Ph.D. (State University of Iowa), ScD. (George Washington), Professor of Zoology, Emeritus, George Washington University; Curator of the Divisions of Mollusks and Cenozoic Invertebrates, *United States National Museum, Washington, D. C.*

Baumgartner, William Jacob, A.B., A.M. (Kansas), Ph.D. (Munich), Professor of Zoology, University of Kansas, *1209 Ohio St., Lawrence, Kan.*

Beach, Frank A., B.S., M.S. (Kansas State Teachers), Ph.D. (Chicago), Chairman and Curator, *Department of Animal Behavior, American Museum of Natural History, Central Park West at 79th St., New York 24, N. Y.*

Beadle, G(eorge) W(ells), B.S., M.S. (Nebraska), Ph.D. (Cornell), Professor of Biology, *Division of Biology, California Institute of Technology, Pasadena 4, Calif.*

Beams, Harold William, A.B. (Fairmount), M.A. (Northwestern), Ph.D. (Wisconsin), Professor of Zoology, *Zoology Department, State University of Iowa, Iowa City, Iowa.*

Bean, Elizabeth Smith, A.B. (Cincinnati), M.A., Ph.D. (Wisconsin), Instructor in Histology and Embryology, *Dahlhousie University, 28 Chestnut St., Halifax, Nova Scotia.*

Beaver, Chester, A.B. (Wabash), M.S., Ph.D. (Illinois), Assistant Professor of Parasitology, *Department of Tropical Medicine, Tulane University School of Medicine, New Orleans 13, La.*

Beccari, Nello, M.D. (Florence), Professor of Comparative Anatomy, *Instituto di Anatomica Comparata. Via Romana, 19, Florence, Italy.*

Beck, Lyle Vibert, A.B. (Wabash College), M.S. (Washington University), Ph.D. (Pittsburgh), Associate Professor of Physiology, *Hahnemann Medical College, Philadelphia, Pa.*

Becker, Elery Ronald, A.B. (Colorado), Sc.D. (Johns Hopkins), Professor of Zoology, *Iowa State College, Ames, Iowa.*

Beckwith, Cora Jipson, B.S. (Michigan), M.A., Ph.D. (Columbia), Professor of Zoology, Emeritus, *Vassar College, Poughkeepsie, N. Y.*

Beers, Charles Dale, A.B., A.M. (North Carolina), Ph.D. (Johns Hopkins), Professor of Zoology, University of North Carolina, *Box 328, Chapel Hill, N. C.*

Behre, Ellinor Helene, A.B. (Radcliffe), Ph.D. (Chicago), Professor of Zoology, Louisiana State University, *Box 8729, University Station, Baton Rouge 3, La.*

Bennett, Harry Jackson, B.S. (Louisiana), M.S., Ph.D. (Illinois), Associate Professor of Zoology and Assistant to the Dean of the College of Arts and Sciences, *Louisiana State University, Baton Rouge, La.* (On leave for military service, '42. Capt., U. S. A.)

Bennitt, Rudolf, S.B., A.M. (Boston), A.M., Ph.D. (Harvard), William Ruckner Professor of Zoology, *106 Wildlife Conservation Building, University of Missouri, Columbia, Mo.*

Berger, Charles Albert, A.B., M.A. (Boston College), Ph.D. (Johns Hopkins), Professor of Cytology, Director of the Biological Laboratory, *Fordham University, New York, N. Y.*

BERTHOLF, LLOYD MILLARD, A.B. (Southwestern), A.M., Ph.D. (Johns Hopkins), Dean of the Faculty and Professor of Biology, *Western Maryland College, Westminster, Md.*

BEUTNER, REINHOLD H., M.D., Ph.D. (Berlin), Professor of Pharmacology, *Hahnemann Medical College, 235 North 15th St., Philadelphia, Pa.*

BEVELANDER, GERRIT, A.B. (Hope), A.M. (Michigan), Ph.D. (Johns Hopkins), Associate Professor of Anatomy, *New York University, College of Dentistry, 477 First Ave., New York 16, N. Y.*

BIGELOW, HENRY B., A.B., A.M., Ph.D. (Harvard), ScD. (Yale), Professor of Zoology, Harvard University; Curator of Oceanography, Museum of Comparative Zoology, *Museum of Comparative Zoology, Cambridge, Mass.*

BIGELOW, MAURICE ALPHEUS, B.S., LL.D. (Ohio Wesleyan), M.S. (Northwestern), Ph.D. (Harvard), Sc.D. (Columbia), Professor of Biology, Emeritus, Teachers College, Columbia University, *525 W. 120th St., New York 27, N. Y.*

BISSONNETTE, THOMAS HUME, M.A. (Queens), Ph.D. (Chicago), Professor of Biology and Head of the Department, *Trinity College, Hartford, Conn.*

BLAKE, CHARLES HENRY, S.B., Ph.D. (Massachusetts Institute of Technology), Assistant Professor of Zoology, *Massachusetts Institute of Technology, Cambridge, Mass.*

BLOOM, WILLIAM, A.B., M.D. (Johns Hopkins), Professor of Anatomy, *Hull Anatomical Laboratory, The University of Chicago, Chicago 37, Ill.*

BLOUNT, RAYMOND FRANK, B.S.A., M.S. (Arizona), Ph.D. (Yale), Professor of Anatomy, *Department of Anatomy, University of Texas, Medical Branch, Galveston, Texas.*

BLUM, HAROLD FRANCIS, A.B., Ph.D. (California), Principal Biophysicist, *Naval Medical Research Institute, National Naval Medical Center, Bethesda, Md.*

BODENSTEIN, DIETRICH, Insect Physiologist, *Medical Research Laboratory, Entomology Section, Edgewood Arsenal, Edgewood, Md.*

BODINE, JOSEPH HALL, A.B., Ph.D. (Pennsylvania), Professor of Zoology, *Zoological Laboratory, State University of Iowa, Iowa City, Iowa.*

BOELL, EDGAR JOHN, B.A. (Dubuque), Ph.D. (Iowa), Associate Professor of Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

BOGART, RALPH, B.S. (Missouri), M.S. (Kansas State), Ph.D. (Cornell), Agent in Animal Husbandry (Cooperative) United States Department of Agriculture and Assistant Professor of Animal Husbandry, *Department of Animal Husbandry, University of Missouri, Columbia, Mo.*

BOOKHOUT, CAZLYN GREEN, A.B. (St. Stephen's), M.A. (Syracuse), Ph.D. (Duke), Assistant Professor of Zoology, *Department of Zoology, Duke University, Durham, N. C.*

BORING, ALICE MIDDLETON, A.B., A.M., Ph.D. (Bryn Mawr), Professor of Biology, *Yenching University, Peiping, China.*

BOZLER, EMIL, Ph.D. (Munich), Associate Professor, *Department of Physiology, Hamilton Hall, Ohio State University, Columbus, Ohio.*

BOYDEN, ALAN ARTHUR, A.B., Ph.D. (Wisconsin), Professor of Zoology, *Rutgers University, New Brunswick, N. J.*

Boyden, Edward Allen, A.B., A.M., Ph.D. (Harvard), Professor of Anatomy and Chairman of the Department, *University of Minnesota, School of Medicine, Minneapolis, Minn.*

Bragg, Arthur Norris, B.S. (Bates), M.A. (Boston University), Ph.D. (Oklahoma), Assistant Professor of Zoology, *Department of Animal Biology, University of Oklahoma, Norman, Okla.*

Brambel, Charles Eugene, A.M., Ph.D. (Johns Hopkins), Instructor in Zoology, *The Johns Hopkins University;* Biochemist, *Mercy Hospital, Baltimore, Md.*

Branch, Hazel E(lizabeth), A.B., A.M. (Kansas), Ph.D. (Cornell), Professor of Zoology, Head of Department, *University of Wichita, Wichita, Kan.*

Brauer, Alfred, A.B. (Kansas), M.A. (Oklahoma), Ph.D. (Chicago), Professor of Zoology, *University of Kentucky, Lexington, Ky.*

Breder, Charles Marcus, Jr., D.Sc. (Newark), Chairman and Curator, Department of Ichthyology, *American Museum of Natural History, Central Park West at 79th St., New York 24, N. Y.*

Breland, Osmond P., B.S. (Mississippi State), Ph.D. (Indiana), Assistant Professor of Zoology, *University of Texas, Austin, Texas.*

Breneman, William Raymond, A.B. (Indiana Central College), Ph.D. (Indiana University), Associate Professor, Waterman Institute and Zoology, Department, *Indiana University, Bloomington, Ind.*

Brinley, Floyd John, B.S. (Colorado State), M.A., Ph.D. (Pennsylvania), Biologist, *U. S. Public Health Service, State Department of Health, Austin, Texas.*

Browman, Ludvig Gustav, B.S., Ph.D. (Chicago), Associate Professor of Zoology, *Department of Zoology, Montana State University, Missoula, Mont.*

Brown, Frank Arthur, Jr., A.B. (Bowdoin), A.M., Ph.D. (Harvard), Associate Professor of Zoology, *Department of Zoology, Northwestern University, Evanston, Ill.*

Brown, Leland Arthur, S.B. (Denison), A.M. (Pittsburgh), Ph.D. (Harvard), Professor of Biology, *Transylvania College, Lexington, Ky.*

Brown, Morden Grant, A.B., A.M., Ph.D. (Stanford), Physicist, Research Division, *Spencer Lens Company, Buffalo 11, N. Y.*

Brues, Charles Thomas, B.S., M.S. (Texas), A.M. (Hon.) (Harvard), Professor of Entomology, Emeritus, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Buchanan, James William, B.S. (Ohio), Ph.D. (Chicago), Professor of Zoology, *Northwestern University, Evanson, Ill.*

Bucksbaum, Ralph, B.S., Ph.D. (Chicago), Assistant Professor of Zoology in the College, Whitman Laboratory of Experimental Zoology, *The University of Chicago, Chicago 37, Ill.*

Buck, John Bonner, A.B., Ph.D. (Johns Hopkins), Cytologist, *Industrial Hygiene Research Laboratory, National Institute of Health, Bethesda 14, Md.*

Budington, Robert Allyn, B.A., M.A., D.Sc. (Williams), Professor of Zoology, Emeritus, Oberlin College, Oberlin, Ohio, *185 Sylvan Blvd., Winter Park, Fla.*

Bullington, Walter Edward, B.S., M.S. (Tennessee), Ph.D. (Kansas), Professor of Biology, *Randolph-Macon College, Ashland, Va.*

Bullock, Theodore Holmes, A.B., Ph.D. (California), Assistant Professor of Anatomy, *Department of Anatomy, University of Missouri School of Medicine, Columbia, Mo.*

Burger, J. Wendell, A.B. (Haverford), A.M. (Lehigh), Ph.D. (Princeton), Assistant Professor of Biology, *Department of Biology, Trinity College, Hartford, Conn.*

Burkenroad, Martin David, Assistant Curator, Bingham Oceanographic Collection, Yale University, *55 Hillhouse Ave., New Haven, Conn.*

Burns, Robert Kyle, Jr., A.B. (Bridgewater), Ph.D. (Yale), Research Associate, *Carnegie Laboratory of Embryology, The Johns Hopkins Medical School. Baltimore 5, Md.*

Burt, Charles Earle, B.S., M.S. (Kansas State), Ph.D. (Michigan), *P.O. Box 536, Topeka, Kan.*

Butcher, Earl Orlo, A.B. (De Pauw), A.M., Ph.D. (Cornell), Associate Professor of Anatomy, *Department of Anatomy, New York University, 477 First Ave., New York City, N. Y.*

Butler, Elmer Grimshaw, A.B., Sc.D. (Syracuse), A.M., Ph.D. (Princeton), Professor of Zoology, *Department of Biology, Princeton University, Princeton, N. J.*

Byerly, Theodore Carroll, A.B., M.S., Ph.D. (Iowa), *Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.*

Byrd, Elon E., B.S., M.S. (Mississippi State College), Ph.D. (Tulane), Associate Professor of Zoology, *Department of Zoology, University of Georgia, Athens, Ga.*

Byrnes, Esther F., B.A., M.A., Ph.D. (Bryn Mawr), *1803 N. Camac St., Philadelphia, Pa.*

Cable, Raymond M., A.B. (Berea), M.S., Ph.D. (New York University), Associate Professor of Zoology, *Purdue University, Lafayette, Ind.*

Calvert, Philip Powell, Ph.D. (Pennsylvania), Professor of Zoology, Emeritus, University of Pennsylvania, *Box 14, Cheyney, Pa.*

Cameron, John Andrew, A.B., A.M. (Nebraska), Ph.D. (Missouri), Associate Professor of Anatomy, Baylor Dental School, *1420 Hall St., Dallas 1, Texas.*

Campbell, Arthur Shackleton, A.B. (Pomona), A.M. (Harvard), Ph.D. (California), Professor of Zoology, *Saint Mary's College, St. Mary's, Calif.*

Carlson, J(ames) Gordon, A.B., Ph.D. (Pennsylvania), Associate Professor of Zoology, *Department of Biology, University of Alabama, University, Ala.*

Carothers, E. Eleanor, A.B., A.M. (Kansas), Ph.D. (Pennsylvania), *134 Ave. C, East, Kingman, Kan.*

Carpenter, Esther, B.A. (Ohio Wesleyan), M.S. (Wisconsin), Ph.D. (Yale), Associate Professor of Zoology, *Smith College, Burton Hall, Northampton, Mass.*

Carroll, Mitchel, B.S., Ph.D. (Pennsylvania), Sc.D. (Franklin and Marshall), Professor and Chairman, Department of Biology, and Chairman Division of Natural Sciences and Mathematics, *Franklin and Marshall College, Lancaster, Pa.*

Carson, Hampton Lawrence, A.B., Ph.D. (Pennsylvania), Instructor in Zoology, *Department of Zoology, Washington University, St. Louis 5, Mo.*

CASPARI, ERNST WOLFGANG, Ph.D. (Goettingen), Assistant Professor of Biology, *Department of Zoology, University of Rochester, Rochester, N. Y.*

CASTEEL, DANA BRACKENRIDGE, A.B. (Allegheny), A.M. (Ohio Wesleyan), Ph.D. (Pennsylvania), Professor of Zoölogy, *University of Texas, Austin, Texas.*

CASTLE, WILLIAM A., B.S. (Denison), Ph.D. (Chicago), Professor of Biology, *Mary Washington College of the University of Virginia, Box 1331, Fredericksburg, Va.*

CASTLE, WILLIAM E., A.B. (Denison), A.M., Ph.D. (Harvard), Research Associate in Genetics, *Hilgard Hall, University of California, Berkeley, Calif.*

CAUSEY, DAVID, A.B. (James Millikin), A.M. (Illinois), Ph.D. (California), Professor of Zoology, *University of Arkansas, Fayetteville, Ark.*

CHALKLEY, HAROLD WILLIAM, B.S. (Mississippi A. and M.), M.A., Ph.D. (Johns Hopkins), Senior Physiologist, *National Cancer Institute, U. S. Public Health Service, Bethesda, Md.*

CHAMBERS, ROBERT, JR., A.B. (Robert), M.A. (Queen's University, Canada), Ph.D. (Munich), Research Professor of Biology, New York University, *Washington Square College, Washington Square, New York, N. Y.*

CHANDLER, DAVID C., A.B. (Greenville), M.A., Ph.D. (Michigan), Associate Professor of Zoology, *Franz Theodore Stone Laboratory, Ohio State University, Put-in-Bay, Ohio.*

CHARIPPER, HARRY ADOLPH, B.S., M.S., Ph.D. (New York), Professor of Biology and Chairman of the Department, *Washington Square College, 100 Washington Square East, New York, N. Y.*

CHARLES, DONALD R., B.S. (Franklin and Marshall), Ph.D. (Columbia), Assistant Professor of Zoology, *Department of Zoology, University of Rochester, Rochester, N. Y.*

CHASE, AURIN MOODY, B.A., M.A. (Amherst), Ph.D. (Columbia), Assistant Professor and Research Associate, *Department of Biology, Princeton University, Princeton, N. J.*

CHASE, HERMAN BURLEIGH, A.B. (Dartmouth), Ph.D. (Chicago), Associate in Zoology, *345 Natural History Building, University of Illinois, Urbana, Ill.*

CHASE, SAMUEL WOOD, A.B. (Bowdoin), A.M., Ph.D. (Harvard), Associate Professor of Histology and Embryology, *Western Reserve University, School of Medicine, 2109 Adelbert Road, Cleveland, Ohio.*

CHEN, TZE-TUAN, B.A. (Fukien Christian University), M.A. (Oberlin), Ph.D. (Pennsylvania), Lecturer in Zoology, *Department of Zoology, University of California at Los Angeles, Los Angeles, Calif.*

CHENEY, RALPH HOLT, B.S., A.M. (Boston), Sc.M., Sc.D. (Harvard), Professor of Biology and Chairman of the Department of Biology; Chairman of the Division of Natural and Physical Sciences, Long Island University, *600 Lafayette Ave., Brooklyn 16, N. Y.*

CHESLEY, L. C., Ph.D. (Duke), Biochemist, *Margaret Hague Maternity Hospital, Jersey City, N. J.*

CHICKERING, ARTHUR MERTON, Ph.B. (Yale), M.S. (Wisconsin), Ph.D. (Michigan), Professor of Biology; Chairman, Division of Science and Mathematics; Chairman, Divisional Council, *Department of Biology, Albion College, Albion, Mich.*

CHILD, CHARLES MANNING, Ph.B., M.S. (Wesleyan), Ph.D. (Leipzig), D.Sc. (Wesleyan), Professor of Zoology, Emeritus, The University of Chicago, *Jordan Hall, Stanford University, Calif.*

CHILD, GEORGE PERCY, B.S., Ph.D. (New York), Assistant Professor, *Amherst College, Amherst, Mass.*

CHURCHILL, EDWARD PERRY, A.B. (Iowa), Ph.D. (Johns Hopkins), Professor of Zoology and Head of the Department, University of South Dakota, *415 South University St., Vermillion, S. D.*

CLARK, LEONARD B., A.B., M.A. (Manitoba), M.A., Ph.D. (Johns Hopkins), Associate Professor of Biology, *Department of Biology, Union College, Schenectady, N. Y.*

CLARKE, GEORGE LEONARD, A.B., A.M., Ph.D. (Harvard), Associate Professor of Zöology, Harvard University, and Marine Biologist, Woods Hole Oceanographic Institution, *Biological Laboratories, Harvard University, Cambridge, Mass.*

CLAUS, PEARL E(LIZABETH), A.B., M.A., Ph.D. (Wisconsin), Research Associate in Zoology, *Biology Building, University of Wisconsin, Madison, Wis.*

CLAUSEN, HARRY JOHN, A.B., M.S. (Iowa), Ph.D. (New York), Assistant Professor of Anatomy, *University of Colorado, School of Medicine, Denver, Colo.*

CLEMENS, WILBERT AMIE, B.A., M.A. (Toronto), Ph.D. (Cornell), Professor of Zoology, *University of British Columbia, Vancouver, British Columbia, Canada.*

CLEVELAND, LEMUEL ROSCOE, B.S. (Mississippi), D.Sc. (Johns Hopkins), Associate Professor of Zoology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

COE, WESLEY R., Ph.B., Ph.D. (Yale), Professor of Biology, Emeritus, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

COKER, ROBERT ERVIN, B.S., M.S. (North Carolina), Ph.D. (Johns Hopkins), Kenan Professor of Zoology, *University of North Carolina, Chapel Hill, N. C.*

COLE, ELBERT CHARLES, A.B. (Middlebury), A.M. (Trinity), Ph.D. (Harvard), Professor of Biology, *Williams College, Williamstown, Mass.*

COLE, LEON J., A.B. (Michigan), Ph.D. (Harvard), Professor of Genetics, University of Wisconsin, *College of Agriculture, University of Wisconsin, Madison, Wis.*

COLE, WILLIAM H(ARDER), A.B. (Hamilton), A.M., Ph.D. (Harvard), Professor of Physiology and Biochemistry, Rutgers University and Director of Research Council, *Rutgers University, New Brunswick, N. J.*

COLIN, EDWARD CECIL, A.B. (Kansas), Ph.D. (Chicago), Professor of Zoology and Genetics, Chicago Teachers College, *6567 Harvard Ave., Chicago, Ill.*

COLLETT, MARY ELIZABETH, A.B. (Wellesley), A.M., Ph.D. (Pennsylvania), Associate Professor of Biology, *Mather College, Western Rserve University, Cleveland, Ohio.*

COLTON, HAROLD SELLERS, B.S., M.A., Ph.D. (Pennsylvania), Professor of Zoology, *University of Pennsylvania, Philadelphia, Pa.*, Director of the Museum of Northern Arizona, *Flagstaff, Ariz.*

CONKLIN, EDWIN GRANT, Ph.D. (Johns Hopkins), D.Sc. (Pennsylvania, Yale), LL.D. (Western Reserve, Johns Hopkins, Pennsylvania), Research Professor of Biology, Emeritus, *Princeton University, Princeton, N. J.*, President, *American Philosophical Society, Independence Square, Philadelphia, Pa.*

Coonfield, Benjamin Randolph, B.S. (Arkansas), M.S. (Kansas State), Ph.D. (Harvard), Associate Professor of Biology, *Department of Biology, Brooklyn College, Bedford Avenue and Avenue H, Brooklyn, N. Y.*

Cooper, Kenneth W., A.B., M.A., Ph.D. (Columbia), Assistant Professor of Biology, *Princeton University, Princeton, N. J.*

Copeland, Manton, S.B., S.M., Ph.D. (Harvard), Professor of Biology, *Department of Biology, Brown University, Providence 12, R. I.*

Copenhaver, Wilfred Monroe, A.B., Ph.D. (Yale), Associate Professor of Anatomy, *College of Physicians and Surgeons, 630 W. 168th St., New York, N. Y.*

Corey, Edward Lyman, A.B. (Western Reserve), Ph.D. (Yale), Assistant Professor of Physiology, *Department of Physiology, School of Medicine, University of Virginia, University, Va.*

Cort, W(illiam) W(alter), A.B. (Colorado College), A.M., Ph.D. (Illinois), Professor of Parasitology, *School of Hygiene and Public Health, The Johns Hopkins University, 615 N. Wolfe St., Baltimore, Md.*

Costello, Donald Paul, A.B. (College of the City of Detroit), Ph.D. (Pennsylvania), Professor of Zoology, *Department of Zoology, University of North Carolina, Chapel Hill, N. C.*

Cowles, Rheinart Parker, A.B. (Stanford), Ph.D. (Johns Hopkins), Professor of Zoology, Emeritus, College of Arts and Sciences, *Department of Biology, The Johns Hopkins University, Baltimore, Md.*

Crabb, Edward D., A.B., M.A. (Oklahoma), Ph.D. (Michigan), Professor of Biology, *Department of Biology, University of Colorado, Boulder, Colo.*

Crampton, Henry Edward, A.B., Ph.D., Sc.D. (Columbia), Professor of Zoology, Emeritus, Columbia University, Research Associate, *American Museum of Natural History, New York, N. Y.*

Creaser, Charles W., A.B., M.S., Ph.D. (Michigan), Professor of Zoology, *Wayne University, Detroit, Mich.*

Creighton, William Steel, B.S. (Roanoke), M.A. (Princeton), M.S., D.Sc. (Harvard), Assistant Professor of Biology, *College of the City of New York, 139th St. and Convent Ave., New York, N. Y.*

Crescitelli, Frederick, Ph.B., Sc.M., Ph.D. (Brown), Acting Assistant Professor in Aviation Medicine, *University of Southern California, Los Angeles 7, Calif.*

Cross, James Cecil, A.B. (Southwestern), M.A., Ph.D. (Texas), Professor of Zoology and Head of the Department of Zoology, *Texas College of Arts and Industries, Kingsville, Texas.*

Curtis, Maynie Rose, A.B., A.M., Ph.D. (Michigan), Associate in Cancer Research, Detroit Institute of Cancer Research, Inc., *Wayne University College of Medicine, Detroit 1, Mich.*

Curtis, Winterton Conway, A.B., A.M. (Williams), Ph.D. (Johns Hopkins), D.Sc. (Williams), Professor of Zoology, University of Missouri, *210 Westmount Ave., Columbia, Mo.*

Dahlgren, Ulric, A.B., M.S. (Princeton), Professor of Biology, Emeritus, *Princeceton University, Princeton, N. J.*

Dalton, Albert Joseph, B.S. (Wesleyan), M.A., Ph.D. (Harvard), Instructor in Biology, *Department of Biology, Brown University, Providence 12, R. I.*

DANFORTH, C(HARLES) H(ASKELL), A.M., Sc.D. (Tufts), Ph.D. (Washington University), Professor of Anatomy, Stanford University, *607 Cabrillo Ave., Stanford University, Calif.*

DARBY, HUGH HACKLAND, B.Sc., M.A., Ph.D. (Columbia), Vice-President and Director of Research, Freezer Foods, Inc., Chicago. Editor-in-chief, American Cookery, New York, *1110 Washington Avenue, Pelham Manor 65, N. Y.*

DAVENPORT, GERTRUDE CROTTY, B.S. (Kansas), *Cold Spring Harbor, Long Island, N. Y.*

DAVIS, DAVID EDWARD, A.B. (Swarthmore), M.A., Ph.D. (Harvard), Research Associate, *Department of Parasitology, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore 5, Md.*

DAVIS, HERBERT SPENCER, Ph.B. (Wesleyan), Ph.D. (Harvard), *Route 16, Box 618, Portland 2, Wash.*

DAWSON, ALDEN BENJAMIN, A.B. (Acadia), Ph.D. (Harvard), D.Sc. (Acadia), Professor of Zoology, *Biological Laboratories, Harvard University, Cambridge 38, Mass.*

DAWSON, JAMES ARTHUR, A.B. (Dalhousie), A.M., Ph.D. (Yale), Professor of Biology, Chairman Department of Biology, *College of the City of New York, 139th St. and Convent Ave., New York, N. Y.*

DEANE, HELEN WENDLER, A.B. (Wellesley), A.M., Ph.D. (Brown), Instructor in Anatomy, *Harvard University Medical School, 25 Shattuck St., Boston 15, Mass.*

DE BRUYN, PETER, P.H., M.D. (Amsterdam), Associate Professor of Anatomy and Chairman of the Department, *Hull Anatomical Laboratory, The University of Chicago, Chicago 37, Ill.*

DEDERER, PAULINE HAMILTON, A.B. (Barnard), A.M., Ph.D. (Columbia), Professor of Zoology, Emeritus, *Connecticut College, New London, Conn.*

DE GARIS, CHARLES FRANCIS, M.D. (Washington), A.B. (Wisconsin), A.M., Ph.D. (Johns Hopkins), F.A.C.P., Professor of Anatomy, *University of Oklahoma, School of Medicine, Oklahoma City, Okla.*

DEMERIC, M(ILISLAV), Ph.D. (Cornell), Director, Department of Genetics, *Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y.*

DEMPSTER, WILFRID TAYLOR, B.S., A.M., Sc.D. (Michigan), Assistant Professor of Anatomy, University of Michigan, Medical School, *Ann Arbor, Mich.*

DILLER, IRENE COREY, A.B. (George Washington), A.M., Ph.D. (Pennsylvania), Research Institute, *The Lankenau Hospital, Philadelphia, Pa.*

DILLER, WILLIAM FREY, A.B. (Franklin and Marshall), Ph.D. (Pennsylvania), Assistant Professor of Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia, Pa.*

DOBZHANSKY, THEODOSIUS, Diploma (Kiev, Russia), Professor of Zoology, *Columbia University, New York, N. Y.*

DODDS, GIDEON S., B.A., M.A. (Colorado), Ph.D. (Pennsylvania), Professor of Histology and Embryology, School of Medicine, West Virginia University, *829 Price St., Morgantown, W. Va.*

DOLLEY, WILLIAM LEE, JR., A.B., A.M. (Randolph-Macon), Ph.D. (Johns Hopkins), Professor of Biology, *University of Buffalo, Buffalo, N. Y.*

Domm, Lincoln V(alentine), A.B. (North Central), Ph.D. (Chicago), Associate Professor of Zoology, *Whitman Laboratory of Experimental Zoology, The University of Chicago, Chicago 37, Ill.*

Doyle, William Lewis, A.M., Ph.D. (Johns Hopkins), Associate Professor of Anatomy, *Hull Anatomical Laboratory, The University of Chicago, Chicago 37, Ill.*

Dreyer, William A., A.B. (Illinois), Ph.D. (Chicago), Associate Professor of Zoology, Department of Zoology, *University of Cincinnati, Cincinnati, Ohio.*

Dunihue, Fred Williams, B.A. (Wabash), M.Sc., Ph.D. (New York University), Associate Professor of Histology and Embryology, *College of Medicine, University of Vermont, Burlington, Vt.*

Dunn, L(eslie) C(larence), S.B. (Dartmouth), S.M., Sc.D. (Harvard), Professor of Zoology, *Columbia University, New York, N. Y.*

DuShane, Graham Phillips, B.A. (Wabash), Ph.D. (Yale), Professor of Biology, *Department of Biology, Stanford University, Palo Alto, Calif.*

Eakin, Richard Marshall, A.B., Ph.D. (California), Associate Professor of Zoology, and Chairman of the *Department of Zoology, University of California, Berkeley, Calif.*

Eastlick, Herbert Leonard, A.B. (Montana), M.S., Ph.D. (Washington University), Assistant Professor of Zoology, *Washington State College, Pullman, Wash.*

Eaton, Theodore Hildreth, Jr., A.B. (Cornell), Ph.D. (California), Assistant Professor of Anatomy, *Department of Anatomy, Georgetown Medical School, Washington 7, D. C.*

Edmondson, Charles Howard, Ph.B., M.S., Ph.D. (Iowa). Professor of Zoology, Emeritus, University of Hawaii, Zoologist, *Bishop Museum, Honolulu 35, Hawaii.*

Eggleton, Frank Egbert, A.B. (Hillsdale), A.M., Ph.D. (Michigan), Associate Professor of Zoology, *University of Michigan, Ann Arbor, Mich.*

Eisenbrandt, Leslie Lee, A.B. (College of Emporia), M.S. (Kansas State), Ph.D. (Rutgers), Assistant Professor of Physiology, Director of Research, *University of Kansas City, School of Dentistry, 10th and Troost Ave., Kansas City, Mo.*

Elias, Hans, Dr.phil. (Giessen), Professor of Histology and Embryology, Middlesex Veterinary School, *103 Riverview Ave., Waltham, Mass.*

Elliott, Alfred M., B.A. (Yankton), M.S., Ph.D. (New York University), Professor of Biology, *State Teachers College, Bemidji, Minn.*

Elliott, Rush, A.B. (Ohio University), A.M. (Ohio State University), Ph.D. (Michigan), Professor of Anatomy, Department of Zoology, Ohio University, *3 Marietta Ave., Athens, Ohio.*

Emerson, Alfred Edwards, B.S., M.A., Ph.D. (Cornell), Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Emerson, Henry Seelye, A.M. (Amherst), Ph.D. (Yale), Assistant Professor of Biology, Amherst College, *58 Seelye St., Amherst, Mass.*

Emery, Frederick Earl, D.V.M., M.S. (Kansas State), Ph.D. (Wisconsin), Associate Professor of Physiology, *Department of Physiology and Pharmacology, University of Arkansas, School of Medicine, Little Rock, Ark.*

Enders, Howard E(dwin), B.S., M.S. (Lebanon Valley), B.S. (Michigan), Ph.D. (Johns Hopkins), Professor of Zoology, Head of the Department of Biology, Dean of the School of Science, Purdue University, *249 Littleton St., West Lafayette, Ind.*

Enders, Robert Kendall, A.B., Ph.D. (Michigan), Professor of Zoology, *Department of Zoology, Swarthmore College, Swarthmore, Pa.*

Engels, William Louis, B.S. (Notre Dame), Ph.D. (California), Associate Professor of Zoology, *Department of Zoology, University of North Carolina, Chapel Hill, N. C.* (On leave for military service, '42. Sgt., U.S.A. Air Force.)

Essex, Hiram E., B.S. (Knox), M.S., Ph.D. (Illinois), Associate, Division of Experimental Surgery and Pathology, Mayo Clinic, Associate Professor of Physiology, Mayo Foundation, University of Minnesota, *Institute of Experimental Medicine, Rochester, Minn.*

Etkin, William, B.S. (College of the City of New York), M.A. (Cornell), Ph.D. (Chicago), Assistant Professor of Biology, *College of the City of New York, Convent Ave. and 139th St., New York, N. Y.*

Evans, Francis Gaynor, B.A. (Coe College), M.A., Ph.D. (Columbia), Instructor in Anatomy, *Department of Anatomy, Wayne University College of Medicine, Detroit 26, Mich.*

Evans, Gertrude, A.B. (Mount Holyoke), Ph.D. (Chicago), Assistant Professor of Biology, *Department of Biology, Wells College, Aurora, N. Y.*

Evans, Llewellyn Thomas, A.B., M.A. (Denver), Ph.D. (Harvard), *3413 Wadsworth St., Wheatridge, Colo.*

Evans, Titus Carr, B.A. (Baylor), M.S., Ph.D. (Iowa), Assistant Professor of Radiology, (Biology), *Radiology Research Laboratory, College of Physicians and Surgeons, Columbia University, New York, N. Y.*

Eversole, Wilburn John, A.B. (Berea College), M.S., Ph.D. (New York), Instructor in Biology, *Department of Biology, The Rice Institute, Houston, Texas.*

Fankhauser, Gerhard, Ph.D. (Berne, Switzerland), Associate Professor of Biology, *Princeton University, Princeton, N. J.*

Farner, Donald S(ankey), B.S. (Hamline), M.A., Ph.D. (Wisconsin), Assistant Professor of Zoology and Assistant Curator of Birds, *Museum of Natural History, University of Kansas, Lawrence, Kan.*

Farris, Edmond J(ohn), B.A. (Buffalo), Ph.D. (Pennsylvania), Executive Director, Associate Member in Anatomy, *The Wistar Institute of Anatomy and Biology, Woodland Ave. and 36th St., Philadelphia 4, Pa.*

Fasten, Nathan, B.S. (College of the City of New York), Ph.D. (Wisconsin), Chief Biologist, State of Washington Pollution Control Commission, *218 Bagley Hall, University of Washington, Seattle 5, Wash.*

Fauré-Fremiet, Emmanuel, Docteur ès Sciences (Paris), Professeur, Directeur du Laboratoire d'Embryogénie Comparée, *Collège de France, Paris V^e, France.*

Fennell, Richard Adams, A.B. (Birmingham Southern), Ph.D. (Johns Hopkins), Associate Professor of Zoology, *Department of Zoology, Michigan State College, East Lansing, Mich.*

Figge, Frank Henry John, A.B. (Colorado College), Ph.D. (Maryland), Associate Professor of Gross Anatomy, *University of Maryland, School of Medicine, Baltimore, Md.*

FINLEY, HAROLD EUGENE, B.S. (Morehouse College), M.S., Ph.D. (Wisconsin), Professor of Biology, *Department of Biology, Morehouse College, Atlanta, Ga.*

FISHER, KENNETH CLARKE, B.A. (Acadia), M.A., Ph.D. (Toronto), Assistant Professor of Animal Physiology, *Department of Zoology, University of Toronto, Toronto 5, Canada.*

FOERSTER, RUSSELL EARLE, B.A., M.A. (British Columbia), Ph.D. (Toronto), Director, Pacific Biological Station, *Nanaimo, British Columbia, Canada.*

FOGG, LLOYD C(LARKE), B.S., M.S. (Dartmouth), Ph.D. (Columbia), *Boston University School of Medicine, 80 E. Concord St., Boston, Mass.*

FOLGER, HARRY THOMAS, A.B. (Indiana), Ph.D. (Johns Hopkins), Research Professor of Biology, *Box 161, Fisk University, Nashville, Tenn.*

FORSTER, ROY PHILIP, B.S. (Marquette), Ph.M., Ph.D. (Wisconsin), Assistant Professor of Zoology, *Dartmouth College, Hanover, N. H.*

FRAPS, RICHARD MASON, B.S., Ph.D. (Chicago), Senior Physiologist, Bureau of Animal Industry, *U. S. Department of Agriculture, Beltsville, Md.*

FRASER, CHARLES MCLEAN, A.B., A.M. (Toronto), Ph.D. (Iowa), LL.D. (British Columbia), F.R.S.C., Professor of Zoology, Emeritus, *University of British Columbia, Vancouver, British Columbia, Canada.*

FRIEDMANN, HERBERT, B.Sc. (College of the City of New York), Ph.D. (Cornell), Curator, Division of Birds, *U. S. National Museum, Smithsonian Institution, Washington, D. C.*

FRIEND, ROGER BOYNTON, B.S. (Massachusetts Agricultural), Ph.D. (Yale), Entomologist, Connecticut Agricultural Experiment Station; Lecturer (Professioral rank) Forest Entomology, Yale University; *123 Huntington St., New Haven, Conn.*

FRIES, ERIK FRITIOF BJERKANDER, A.B., A.M., Ph.D. (Harvard), Assistant Professor of Biology, College of the City of New York, *248 West 17th St., New York, N. Y.* (On leave for military service, '42. Lt. U. S. N. R.) *South Brewster, Mass.*

FRISCH, JOHN A., S.J., A.B., M.A. (Woodstock), M.S. (Canisius), Ph.D. (Johns Hopkins), Professor of Biology, Head of the Department, *Canisius College, Buffalo, N. Y.*

FULLER, JOHN LANGWORTHY, B.S. (Bates), Ph.D. (Massachusetts Institute of Technology), Associate Professor of Zoology, *25 Coburn Hall, University of Maine, Orono, Me.*

FULTON, JOHN FARQUHAR, B.S., M.D. (Harvard), B.A., M.A., D.Phil. (Oxford), M.A. (Hon.) (Yale), LL.D. (Hon.) (Kenyon), D.Sc. (Oxford), Sterling Professor of Physiology, Yale University School of Medicine, *333 Cedar St., New Haven, Conn.*

FUNKHOUSER, WILLIAM DELBERT, A.B. (Wabash), M.A., Ph.D. (Cornell), Sc.D. (Wabash), Professor of Zoology and Dean, Graduate School, *University of Kentucky, Lexington, Ky.*

GALTSOFF, PAUL S., Graduate (Moscow), Ph.D. (Columbia), Senior Biologist, *U. S. Fish and Wildlife Service, College Park, Md.*

GARDINER, MARY SUMMERFIELD, A.B., M.A., Ph.D. (Bryn Mawr), Associate Professor of Biology, Bryn Mawr College, *Dalton Hall, Bryn Mawr, Pa.*

Garman, Harrison, D.Sc. (Kentucky), Professor of Entomology, Emeritus, University of Kentucky, *638 South Limestone St., Lexington, Ky.*

Gates, William Hazen, A.B. (Williams), D.Sc. (Harvard), Professor of Zoology and Entomology, *Louisiana State University, Baton Rouge 3, La.*

Gaunt, Robert, B.A. (Tulsa), M.A., Ph.D. (Princeton), Professor of Zoology and Chairman of the Department, *Syracuse University, Syracuse, N. Y.*

Geiser, Samuel Wood, B.A., M.A. (Upper Iowa), Ph.D. (Johns Hopkins), Professor of Biology, *Southern Methodist University, Dallas, Texas.*

Geiman, Quentin Monroe, B.S. (Gettysburg College), M.S., Ph.D. (Pennsylvania), Assistant Professor of Tropical Diseases, *Department of Comparative Pathology and Tropical Medicine, Harvard Medical School and School of Public Health, Boston 15, Mass.*

Gerould, John Hiram, Litt.B. (Dartmouth), A.B., A.M., Ph.D. (Harvard), Professor of Zoology, Emeritus, Dartmouth College, *36 Occom Ridge, Hanover, N. H.*

Giese, Arthur Charles, B.S. (Chicago), Ph.D. (Stanford), Associate Professor of Biology, *Stanford University, Calif.*

Gilbert, Chauncey McLean, B.S. (Massachusetts), Ph.D. (Virginia), Assistant Professor of Biology, University of Virginia, *3 Rugby Apartments, University Station, Charlottesville, Va.*

Gilchrist, Francis Garritt, A.B., Ph.D. (California), *Riverside Junior College, Riverside, Calif.*

Glaser, Otto Charles, A.B. (Johns Hopkins), A.M. (Amherst), Ph.D. (Johns Hopkins), Harkness Professor of Biology, *Amherst College, Amherst, Mass.*

Glass, H(iram) Bentley, A.B., M.A. (Baylor), Ph.D. (Texas), Professor of Biology, *Goucher College, Baltimore, Md.*

Gluecksohn-Schoenheimer, Salome, Ph.D. (Freiburg i. Br.), Research Associate, *Department of Zoology, Columbia University, New York, N. Y.*

Goldforb, A. J., B.S. (College of the City of New York), Ph.D. (Columbia), General Secretary and Editor in Chief, Proceedings Society for Experimental Biology and Medicine, Professor of Biology, *College of the City of New York, New York, N. Y.*

Goldschmidt, Richard B., Ph.D. (Heidelberg), M.D. (Kiel), Sc.D. (Madrid), Professor of Zoology, *Department of Zoology, University of California, Berkeley, Calif.*

Goldsmith, Eli D., B.S. (College of the City of New York), M.S. (New York), M.A., Ph.D. (Harvard), Instructor in Biology, *College of Dentistry, New York University, 477 First Ave., New York, N. Y.*

Goldsmith, Joseph B(enjamin), A.B. (Southwestern), A.M. (Oklahoma), Ph.D. (Wisconsin), Associate Professor, Department of Histology and Embryology, *Oklahoma University School of Medicine, Oklahoma City, Okla.*

Goldsmith, William Marion, B.Pd., A.B. (Hillsdale), A.M., Ph.D. (Indiana), Instructor in Zoology, *Department of Zoology, University of Dubuque, Iowa.*

Goodale, Hubert Dana, Ph.D. (Columbia), Biologist, Mount Hope Farm, *557 W. Main St., Williamstown, Mass.*

GOODNIGHT, CLARENCE JAMES, A.B., M.A., Ph.D. (Illinois), Assistant Professor of Zoology, *Department of Biology, Purdue University, West Lafayette, Ind.*

GOODRICH, HUBERT BAKER, B.S. (Amherst), M.A., Ph.D. (Columbia), Professor of Biology, *Wesleyan University, Middletown, Conn.*

GORBMAN, AUBREY, A.B., M.S. (Wayne), Ph.D. (California), Assistant Professor of Zoology, *Department of Zoology, Barnard College, Columbia University, New York 27, N. Y.*

GORDON, ALBERT S(AUL), B.S. (College of the City of New York), M.S., Ph.D. (New York), Assistant Professor of Biology, *Department of Biology, Washington Square College, New York University, New York, N. Y.*

GORDON, MYRON, B.S., Ph.D. (Cornell), Research Associate in Genetics of the New York Aquarium, New York Zoological Society, *American Museum of Natural History, New York 24, N. Y.*

GOWEN, JOHN WHITTEMORE, B.S. (Maine), M.S., Ph.D. (Columbia), Professor of Genetics, *Iowa State College, Ames, Iowa.*

GRAHAM, GEORGE LAURIN, A.B. (Grand Island), M.S. (Kansas State), Sc.D. (Johns Hopkins), Associate, Department of Animal and Plant Pathology, *Rockefeller Institute for Medical Research, Princeton, N. J.* (On leave for military service, '44, Capt. Sanitary Corps. U.S.A.)

GRAHAM, JOHN YOUNG, Ph.D. (Munich), Professor of Biology, *University of Alabama, University, Ala.*

GRAY, IRVING E., B.S. (Massachusetts State), M.S., Ph.D. (Wisconsin), Professor of Zoology, *Duke University, Durham, N. C.*

GRAY, JAMES CLARKE, A.B., M.A. (Syracuse), Ph.D. (Chicago), Associate Professor of Biology, *Western Reserve University, Cleveland, Ohio.*

GRAY, PETER, Ph.D. (London), D.I.C. Professor of Biology, *University of Pittsburgh, Pittsburgh, Pa.*

GREEP, ROY ORVAL, B.S. (Kansas State Agricultural College), M.S., Ph.D. (Wisconsin), Assistant Professor of Dental Science, Harvard School of Dental Medicine, and Teaching Fellow, *Department of Anatomy, Harvard Medical School, 25 Shattuck St., Boston, Mass.*

GREGG, ROBERT E., S.B., Ph.D. (Chicago), Instructor in Zoology, *Department of Biology, University of Colorado, Boulder, Colo.*

GREGORY, LOUISE HOYT, A.B. (Vassar), A.M., Ph.D. (Columbia), Professor of Zoology and Associate Dean, *Barnard College, New York, N. Y.*

GRIFFEN, ALLEN BEATTIE, B.A., Ph.D. (Texas), Associate Professor of Zoology, *Department of Zoology, University of Texas, Austin, Texas.*

GRIFFIN, LAWRENCE EDMONDS, A.B., Ph.B. (Hamline), Ph.D. (Johns Hopkins), Professor of Biology, *Reed College, Portland, Ore.*

GROBMAN, ARNOLD BRAMS, B.S. (Michigan), M.S., Ph.D. (Rochester), Assistant Professor of Biology, *Department of Biology, University of Florida, Gainsville, Fla.*

GUDGER, E. W., B.S., M.S. (Nashville), Ph.D. (Johns Hopkins), Honorary Associate Librarian, Dean Memorial Library, *American Museum of Natural History, West 77th St., New York 24, N. Y.*

GUHL, ALPHAEUS MATTHEW, B.A. (North Central), M.S., Ph.D. (Chicago), Instructor in Zoology, *Department of Zoology, Kansas State College, Manhattan, Kan.*

GULICK, ADDISON, A.B. (Oberlin), A.M. (Harvard), Ph.D. (Würzburg, Germany), Professor of Physiological Chemistry, University of Missouri, *308 Westmount Ave., Columbia, Mo.*

GUTHRIE, MARIE JANE, A.B., A.M. (Missouri), Ph.D. (Bryn Mawr), Professor of Zoology, University of Missouri, *Lefevre Hall, University of Missouri, Columbia, Mo.*

GUYER, MICHAEL FREDERIC, B.S. (Chicago), A.M. (Nebraska), Ph.D. (Chicago), LL.D. (Missouri), Professor of Zoology, *Biology Building, University of Wisconsin, Madison, Wis.*

HADLEY, CHARLES ELMER, B.A. (Bates), M.A. (Trinity, Harvard), Ph.D. (Harvard), Associate Professor of Biology, *New Jersey State Teachers College, Upper Montclair, N. J.*

HAGUE, FLORENCE SANDER, B.A., M.A. (Kansas), Ph.D. (Illinois), Associate Professor of Biology, *Sweet Briar College, Sweet Briar, Va.*

HAHNERT, WILLIAM F(RANKLIN), A.B. (De Pauw), Ph.D. (Johns Hopkins), Associate Professor and Acting Head of the *Department of Zoology, Ohio Wesleyan University, Delaware, Ohio.*

HAIG, CHARLES, B.S., M.A., Ph.D. (Columbia), Assistant Professor of Physiology and Biochemistry, *New York Medical College, 5th Ave. and 106th St., New York, N. Y.*

HALL, EDMUND KENNARD, B.S. (Princeton), Ph.D. (Yale), D.Sc. (Brussels), Assistant Professor of Anatomy, School of Medicine, University of Louisville, *101 W. Chestnut St., Louisville, Ky.*

HALL, E(UGENE) RAYMOND, A.B. (Kansas), M.A., Ph.D. (California), Professor of Zoology, Chairman of the Department of Zoology, and Director of the Museum of Natural History, *University of Kansas, Lawrence, Kan.*

HALL, FRANK GREGORY, B.A. (Milton), M.A., Ph.D. (Wisconsin), Professor of Physiology, *Duke University, Durham, N. C.*

HALL, RICHARD P., A.B. (Henderson-Brown), A.M., Ph.D. (California), Professor of Biology, *New York University, University Heights, N. Y.*

HAMAKER, JOHN IRVIN, A.B. (Kansas), A.B., A.M., Ph.D. (Harvard), Professor of Biology, Randolph-Macon Woman's College, *16 N. Princeton St., Lynchburg, Va.*

HAMBURGER, VIKTOR, Ph.D. (Freiburg), Professor of Zoology, *Washington University, St. Louis, Mo.*

HAMILTON, HOWARD LAVERNE, B.A., M.S. (Iowa State), Ph.D. (Johns Hopkins), Assistant Professor of Zoology, *Department of Zoology and Entomology, Iowa State College, Ames, Iowa.*

HAMILTON, WILLIAM JOHN, JR., B.S., M.S., Ph.D. (Cornell), Associate Professor of Zoology, *Department of Zoology, Cornell University, Ithaca, N. Y.*

HAMLETT, GEORGE WHITFIELD DELUZ, B. A., M.A., Ph.D. (Texas), Agent, U. S. Biological Survey, *404 E. 20th St., Baltimore, Md.*

HAMMETT, FREDERICK SIMONDS, A.B. (Tufts), M.S. (Rhode Island State Agricultural College), A.M., Ph.D. (Harvard), Scientific Director, The Lankenau Hospital Research Institute, Philadelphia, Pa. *493 Commercial St., Provincetown, Mass.*

HAMRE, CHRISTOPHER JOHN, B.A. (St. Olaf), M.S., Ph.D. (Wisconsin), Professor of Zoology and Chairman of the Department, *University of Hawaii, Honolulu, Hawaii.*

HANCE, ROBERT THEODORE, A.B., M.A. (Cincinnati), Ph.D. (Pennsylvania), Vice-President, Rosco Laboratories, Inc., Blawnox, Pa., Research Biologist, *Cincinnati Milling Machine Co., Cincinnati, Ohio.*

HANN, HARRY WILBUR, A.B. (Indiana), A.M., Ph.D. (Michigan), Assistant Professor of Zoology, *University of Michigan, Ann Arbor, Mich.*

HARD, WALTER LEON, A.B. (Albion), Ph.D. (Duke), Assistant Professor of Anatomy, *Department of Anatomy, Medical College of the State of South Carolina, Charleston 16, S. C.*

HARDESTY, MARY (Mrs. W. L. Duren, Jr.), B.A. (Tulane), M.S., Ph.D. (Chicago), Acting Instructor in Zoology, Tulane University, *3323 Joseph St., New Orleans, La.*

HARGITT, GEORGE THOMAS, Ph.B., Sc.D. (Syracuse), A.M. (Nebraska), Ph.D. (Harvard), Professor of Zoology, *Biology Building, Duke University, Durham, N. C.*

HARMAN, MARY THERESA, A.B., M.A., Ph.D. (Indiana), Professor of Zoology, *Kansas State College, Manhattan, Kan.*

HARRIS, MORGAN, A.B., Ph.D. (California), Assistant Professor of Zoology, *Department of Zoology, University of California, Berkeley, Calif.*

HARRISON, JAMES A(LEXANDER), A.B. (Trinity), Ph.D. (Chicago), Professor of Biology, *Department of Biology, Temple University, Philadelphia 22, Pa.*

HARRISON, ROSS GRANVILLE, Ph.D. (Johns Hopkins), M.D. (Bonn), Sc.D. (Cincinnati, Michigan, Dublin, Harvard, Yale, Columbia), Ph.D. (Freiburg), M.D. (Budapest), LL.D. (Johns Hopkins), Sterling Professor of Biology, Emeritus, Yale University; Chairman, National Research Council, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

HARTMAN, CARL G., Ph.D. (Texas), Professor of Zoology, Head of Department of Zoology and Physiology and Chairman of Biological Division, *University of Illinois, Urbana, Ill.*

HARTMAN, FRANK ALEXANDER, A.B., A.M. (Kansas), Ph.D. (Washington), Professor of Physiology and Chairman of the Department, *Hamilton Hall, Ohio State University, Columbus, Ohio.*

HARVEY, EDMUND NEWTON, Ph.D. (Columbia), Henry Fairfield Osborn Professor of Biology, *Princeton University, Princeton, N. J.*

HARVEY, ETHEL BROWNE, Ph.D. (Columbia), Princeton University, *48 Cleveland Lane, Princeton, N. J.*

HASKINS, CARYL PARKER, Ph.B. (Yale), Ph.D. (Harvard), *Greenacres, Green Acre Lane, Westport, Conn.*

HASLER, ARTHUR D., B.A. (Brigham Young), Ph.D. (Wisconsin), Associate Professor of Zoology, *Biology Building, University of Wisconsin, Madison, Wis.*

HATCH, MELVILLE HARRISON, A.B., M.A., Ph.D. (Michigan), Professor of Zoology, *University of Washington, Seattle, Wash.*

HATERIUS, HANS OLIVER, A.B. (Augustana), M.S., Ph.D. (Iowa), Professor of Physiology, *Boston University School of Medicine, 80 East Concord, Boston 18, Mass.*

HATT, ROBERT, T., B.S. (Michigan), M.A., Ph.D. (Columbia), Director, Cranbrook Institute of Science, *Bloomfield Hills, Mich.*

HAUSCHKA, THEODORE SPAETH, A.B. (Princeton), M.S., Ph.D. (Pennsylvania), Biologist, *Lankenau Hospital Research Institute, Girard and Corinthian Aves., Philadelphia 30, Pa.*

HAYDEN, MARGARET ALGER, A. B., A.M., Ph.D. (Columbia), Professor of Zoology, *Wellesley College, Wellesley, Mass.*

HAYS, FRANK ALFRED, B.S. (Oklahoma), M.A. (Nebraska), Ph.D. (Iowa State), Research Professor in Poultry Investigations, *Massachusetts Agricultural Experiment Station, Amherst, Mass.*

HECHT, SELIG, B.S. (College of the City of New York), Ph.D. (Harvard), Professor of Biophysics, Columbia University, *35 Claremont Ave., New York, N. Y.*

HEILBRUNN, LEWIS VICTOR, A.B., Ph.D. (Chicago), Professor of Zoology, *Zoological Laboratory, University of Pennsylvania, Philadelphia 4, Pa.*

HELFF, OTTO MAXIMILIAN, B.S. (New Hampshire), M.S. (Chicago), Ph.D. (Yale), Associate Professor of Biology, *New York University, University Heights, New York, N. Y.*

HELLBAUM, ARTHUR ALFRED, B.A. (St. Olaf), M.A., Ph.D. (Wisconsin), M.D. (Chicago), Professor of Pharmacology, *Oklahoma University, School of Medicine, Oklahoma City, Okla.*

HELWIG, EDWIN ROYER, A.B. (Western Maryland), Ph.D. (Pennsylvania), Assistant Professor of Zoology, *University of Pennsylvania, Philadelphia, Pa.*

HENSHAW, PAUL STEWART, A.B. (Southwestern College), M.S., Ph.D. (Wisconsin), Biophysicist at Memorial Hospital, *2 W. 106th St., New York, N. Y.*

HERRICK, CHARLES JUDSON, Ph.D. (Columbia), Professor of Neurology, Emeritus, The University of Chicago, *236 Morningside Drive, Grand Rapids 6, Mich.*

HERRICK, CHESTER ALBERN, B.S., M.S. (Kansas State College), Sc.D. (Johns Hopkins), Professor of Zoology and of Veterinary Science, *Department of Zoology, University of Wisconsin, Madison, Wis.*

HERRICK, EARL H., B.S., M.S. (Kansas State College), Ph.D. (Harvard), Professor of Zoology and Experiment Station Mammalogist, *Department of Zoology, Kansas State College, Manhattan, Kan.*

HERSH, AMOS HENRY, A.B. (Franklin and Marshall), Ph.D. (Illinois), Associate Professor of Biology, *Western Reserve University, Cleveland, Ohio.*

HESS, WALTER NORTON, A.B. (Oberlin), A.M., Ph.D. (Cornell), Professor of Biology, *Department of Biology, Hamilton College, Clinton, N. Y.*

HEUSTIS, RALPH RUSKIN, B.S.A. (McGill), M.S., Ph.D. (California), Professor of Zoology, *University of Oregon, Eugene, Ore.*

HIBBARD, HOPE, A.B., A.M. (Missouri), Ph.D. (Bryn Mawr), Sc.D. (Sorbonne), Professor of Zoology, *Oberlin College, Oberlin, Ohio.*

HIESTAND, WILLIAM ANDREW, B.A., M.A., Ph.D. (Wisconsin), Associate Professor of Physiology, Purdue University, *Hillcrest Rd., West Lafayette, Ind.*

HILE, RALPH, A.B. (Indiana Central), Ph.D. (Indiana), Associate Aquatic Biologist, U. S. Fish and Wildlife Service, *1027 University Museum, Ann Arbor, Mich.*

HILL, SAMUEL E., B.S. (Texas Christian), A.M., Ph.D. (Princeton), Professor of Biology, *Russell Sage College, Troy, N. Y.*

Hilton, William Atwood, B.S., Ph.D. (Cornell), Professor of Zoology, Pomona College; Director Laguna Marine Biological Laboratory, Laguna Beach, Calif., *1263 Dartmouth St., Claremont, Calif.*

Hisaw, Frederick Lee, A.B., B.S., A.M., LL.D. (Missouri), Ph.D. (Wisconsin), Professor of Zoology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Hitchcock, Fred Andrews, M.S., Ph.D. (Ohio State), Professor of Physiology, *Ohio State University, Columbus, Ohio.*

Hoadley, Leigh, A.B. (Michigan), Ph.D. (Chicago), Professor of Zoology, Biological Laboratories, *Harvard University, Cambridge, Mass.*

Hoagland, Hudson, A.B. (Columbia), M.S. (Massachusetts Institute of Technology), Ph.D. (Harvard), Executive Director, Worcester Foundation for Experimental Biology. Fellow, John Simon Guggenheim Memorial Foundation. *Box 57, Worcester, Mass.*

Hobbs, Elizabeth Sanders, (Mrs. Joseph R.), A.B. (Goucher), D.Sc. (Johns Hopkins), Assistant Professor of Zoology, *Smith College, Northampton, Mass.*

Hodge, Charles 4th, A.B., Ph.D. (Pennsylvania), Assistant Professor, *Department of Biology, Temple University, Philadelphia, Pa.*

Hoff, C(larence) Clayton, A.B. (Bradley), M.S., Ph.D. (Illinois), Assistant Professor of Zoology, *Department of Zoology and Parasitology, Veterinary Division, Colorado Agricultural and Mechanical College, Fort Collins, Colo.*

Hogue, Mary Jane, A.B. (Goucher), Ph.D. (Würzburg), Associate in Anatomy, *University of Pennsylvania, School of Medicine, Philadelphia, Pa.*

Hollander, Willard Fisher, B.A. (Texas), M.S., Ph.D. (Wisconsin), Instructor in Biology, *Larson Junior College, 1450 Whitney Ave., New Haven 14, Conn.*

Holmes, Samuel Jackson, B.S., M.S. (California), Ph.D. (Chicago), Professor of Zoology, *University of California, Berkeley, Calif.*

Holmquist, Albert M., A.B. (St. Olaf), M.S., Ph.D. (Chicago), Professor of Biology, *St. Olaf College, Northfield, Minn.*

Hooker, Charles Wright, A.B., A.M., Ph.D. (Duke), Assistant Professor of Anatomy, *Yale University School of Medicine, New Haven, Conn.*

Hopkins, Dwight Lucian, B.S., M.S. (Virginia), Ph.D. (Johns Hopkins), Professor of Zoology, *Mundelein College, Chicago, Ill.*

Hopkins, Hoyt S., A.B., A.M. (Oberlin), Ph.D. (Hopkins), Associate Professor of Physiology, New York University, College of Dentistry, *209 E. 23rd St., New York, N. Y.*

Hopkins, Sewell Hepburn, B.S. (William and Mary), M.A., Ph.D. (Illinois), Biologist, *Virginia Fisheries Laboratory, Yorktown, Va.*

Houser, Gilbert Logan, B.S., M.S. (Iowa), Ph.D. (Johns Hopkins), Professor of Zoology, State University of Iowa, *430 Iowa Ave., Iowa City, Iowa.*

Hoy, William E., A.B. (Franklin and Marshall), Ph.D. (Princeton), Professor of Biology and Head of the Department, *University of South Carolina, Columbus, S. C.*

Hubbs, Carl L(eavitt), A.B., A.M. (Stanford), Ph.D. (Michigan), Curator of Fishes; Professor of Zoology; *Scripps Institution of Oceanography, University of California, La Jolla, Calif.*

Huettner, Alfred F., A.B. (South Dakota), A.M., Ph.D. (Columbia), Associate Professor of Biology, *Queens College, Flushing, N. Y.*

HUGHES-SCHRADER, SALLY, B.S. (Grinnell), Ph.D. (Columbia), *Department of Zoology, Columbia University, New York, N. Y.*

HULL, FRANK MONTGOMERY, B.S. (Mississippi State College), M.S. (Ohio State), Ph.D. (Harvard), Professor of Zoology, *Department of Biology, University of Mississippi, University, Miss.*

HULPIEU, HAROLD RAYMOND, A.B. (Southwestern), M.A. (Oklahoma), Ph.D. (Johns Hopkins), Associate Professor, Department of Pharmacology, *Indiana University School of Medicine, Indianapolis, Ind.*

HUMM, FRANCIS DORRIS, A.B. (Smith), M.A., Ph.D. (Yale), Sigma Delta Epsilon Research Fellow in Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

HUMPHREY, RUFUS RICHARD, A.B. (Michigan State Normal), A.M., Ph.D. (Cornell), Professor of Anatomy, *University of Buffalo, 24 High St., Buffalo, N. Y.*

HUNGATE, ROBERT EDWARD, A.B., Ph.D. (Stanford), Associate Professor of Zoology, and Research Biologist, The Biochemical Institute, *Department of Zoology and Physiology, The University of Texas, Austin, Texas.*

HUNT, HARRISON RANDALL, B.S. (Allegheny), M.A., Ph.D. (Harvard), Head of the Department of Zoology and Zoologist in the Agricultural Experiment Station, Michigan State College, East Lansing, Mich., *1050 North Hagadorn Rd., East Lansing, Mich.*

HUNT, THOMAS ELWOOD, A.B. (Earlham), Ph.D. (Chicago), Associate Professor of Histology and Embryology, *Medical College of Alabama, Birmingham 5, Ala.*

HUNTER, FRANCIS ROBERT, B.S. (California Institute of Technology), M.A. (Wesleyan), Ph.D. (Princeton), Assistant Professor of Physiology, Department of Animal Biology, *University of Oklahoma, Norman, Okla.*

HUNTER, GEORGE WILLIAM, A.B., A.M. (Williams), Ph.D. (New York), Lecturer in Science Education, Graduate School, Associated Colleges at *Claremont, Claremont, Calif.*

HUNTER, GEORGE WILLIAM, III, B.S. (Knox), M.S., Ph.D. (Illinois), Major, Sn.C., Assistant Chief, Division of Parasitology, *Army Medical School, Army Medical Center, Washington 12, D. C.*

HUNTSMAN, ARCHIBALD GOWANLOCK, B.A., M.D. (Toronto), F.R.S.C. (Canada), Professor of Marine Biology, Editor and Consulting Director, Fisheries Research Board of Canada, *University of Toronto, Toronto, Canada.*

HURST, CLARENCE THOMAS, A.B., A.M. (Western State College of Colorado), Ph.D. (California), Professor of Zoology and Dean of the Graduate School, *Western State College of Colorado, Gunnison, Colo.*

HUSSAKOFF, LOUIS, B.S. (College of the City of New York), Ph.D. (Columbia), Research Associate, Department of Ichthyology, *American Museum of Natural History, 79th St. and Central Park West, New York 24, N. Y.*

HUTCHINSON, CRANFORD, A.B. (Ohio Wesleyan), Ph.D. (Yale), Instructor in Anatomy, *Jefferson Medical College, Philadelphia, Pa.*

HUTCHINSON, G(EORGE) EVELYN, B.A., M.A. (Cambridge), Associate Professor of Zoology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

HUTT, FREDERICK BRUCE, B.S.A. (Toronto), M.S. (Wisconsin), M.A. (Manitoba), Ph.D., D.Sc. (Edinburgh), Professor of Animal Genetics, *Cornell University, Ithaca, N. Y.*

Hyman, Libbie Henrietta, B.S., Ph.D., Sc.D. (Chicago), Research Associate, Department of Invertebrates, *American Museum of Natural History, New York, N. Y.*

Ibsen, Heman Lauritz, B.S., M.S., Ph.D. (Wisconsin), Professor of Genetics, Department of Animal Husbandry, *Kansas State College, Manhattan, Kan.*

Illick, John Theron, A.B., A.M. (Syracuse), Ph.D. (Princeton), Associate Professor of Zoology, *Lyman Hall, Syracuse University, Syracuse, N. Y.*

Ingles, Lloyd Glenn, A.B. (Redlands), M.A. (Claremont), Ph.D. (California), Professor of Biology, *Chico State College, Chico, Calif.*

Irving, Laurence, A.B. (Bowdoin), A.M. (Harvard), Ph.D. (Stanford), Professor of Biology and Chairman of the Department of Zoology, *Swarthmore College, Swarthmore, Pa.*

Irwin, Malcolm Robert, B.S., M.S., Ph.D. (Iowa State College), Professor of Genetics, *Department of Genetics, University of Wisconsin, Madison, Wis.*

Isely, Frederick B., B.S. (Wichita), M.S. (Chicago), Professor of Biology, *Trinity University, San Antonio, Texas.*

Ives, Philip Truman, A.B., M.A. (Amherst), Ph.D. (California Institute of Technology), Research Associate in Biology, *Department of Biology, Amherst College, Amherst, Mass.*

Jacobs, Merkel Henry, A.B., Ph.D. (Pennsylvania), Professor of General Physiology, *University of Pennsylvania, Philadelphia, Pa.*

Jahn, Theodore Louis, B.A. (Rice Institute), M.Sc., Ph.D. (New York), Associate Professor, Department of Zoology, *State University of Iowa, Iowa City, Iowa.*

Janes, Ralph Gordon, A.B. (Battle Creek), M.S., Ph.D. (New York), Assistant Professor of Anatomy, *State University of Iowa, Iowa City, Iowa.*

Jeffers, Katharine R., A.B., A.M. (Missouri), Ph.D. (Bryn Mawr), Assistant Professor of Zoology, *Duke University, Durham, N. C.*

Jennings, Herbert Spencer, B.S. (Michigan), A.M., Ph.D. (Harvard), LL.D. (Clark, Pennsylvania), Sc.D. (Michigan, Chicago), LL.D. (California), Sc.D. (Oberlin), Professor of Zoology, Emeritus, The Johns Hopkins University, *Department of Zoology, University of California, Los Angeles, Calif.*

Jewell, Minna Ernestine, A.B. (Colorado), A.M., Ph.D. (Illinois), Department of Biology, *Thornton Junior College, Harvey, Ill.*

Job, Thesle T., A.B. (Simpson), M.S., Ph.D. (Iowa), Professor of Anatomy, Loyola University School of Medicine, *706 S. Wolcott St., Chicago 12, Ill.*

Johannsen, Oskar Augustus, B.S. (Illinois), A.M., Ph.D. (Cornell), Professor of Entomology, Emeritus, College of Agriculture, *Cornell University, Ithaca, N. Y.*

Johnson, Frank Harris, A.B. (Princeton), A.M. (Duke), Ph.D. (Princeton), Assistant Professor of Biology, *Department of Biology, Princeton University, Princeton, N. J.*

Johnson, Henry Herbert, B.S. (Mercer), A.M., Ph.D. (Columbia), Associate Professor of Biology, *Department of Biology, College of the City of New York, New York, N. Y.*

Johnson, Percy Leroy, B.S. (Maine), M.S. (Syracuse), Ph.D. (Johns Hopkins), Assistant Professor of Anatomy, *College of Dentistry, New York University, 477 First Ave., New York, N. Y.*

Johnson, Willis Hugh, A.B. (Wabash), M.S., Ph.D. (Chicago), Professor of Zoology and Chairman of the Department, *Wabash College, Crawfordsville, Ind.*

Jones, E. Elizabeth, A.B., Ph.D. (Radcliffe), A.M. (Maine), Associate Professor of Zoology, *Wellesley College, Wellesley, Mass.*

Jones, E(dmund) Ruffin, Jr., B.A., B.S., M.A., Ph.D. (Virginia), Professor of Biology, *College of William and Mary, Norfolk Division, Norfolk, Va.*

Jones, Sarah Von Hoosen, Ph.B. (Chicago), M.S., Ph.D. (Wisconsin), *Rochester, Mich.*

Jordan, Harvey Ernest, B.A., M.A., Sc.D. (Hon.) (Lehigh), Ph.D. (Princeton), Professor of Anatomy and Director of the Anatomical Laboratories; Dean, Department of Medicine, *Pavilion 8, East Lawn, University of Virginia, University, Va.*

Juhn, Mary, Ph.D. (Zurich), Research Professor, *Department of Poultry Husbandry, University of Maryland, College Park, Md.*

Kaston, B(enjamin) J(ulian), B.S. (North Carolina State College), Ph.D. (Yale), Professor of Zoology, *Department of Zoology, Syracuse University, Syracuse, N. Y.*

Kaufmann, Berwind P(etersen), B.S., M.A., Ph.D. (Pennsylvania), Resident Investigator, Department of Genetics, *Carnegie Institution of Washington, Cold Spring Harbor, N. Y.*

Keeler, Clyde Edgar, B.S., M.S. (Denison), M.A., D.Sc. (Harvard), Instructor in Anatomy, Harvard Medical School, *Shattuck St., Boston Mass.* (On leave for military service, '43.)

Kempton, Rudolf T., B.S. (Bates), M.A. (Columbia), Ph.D. (New York), Professor of Zoology, Chairman of the Department of Zoology, and Curator of the Natural History Museum, *Vassar College, Poughkeepsie, N. Y.*

Kendall, James I., B.S. (New York), M.S., Ph.D. (Harvard), D.Sc. (Sofia), Assistant Professor of Biology, College of the City of New York, *P.O. 14, Tenafly, N. J.*

Kendeigh, S. Charles, A.B., A.M. (Oberlin), Ph.D. (Illinois), Associate Professor of Zoology, University of Illinois, Vivarium Building, *Wright and Healey Sts. Champaign, Ill.*

Kenk, Roman, Ph.D. (Graz), Austria), Professor of Biology, *Department of Biology, University of Puerto Rico, Rio Piedras, Puerto Rico.*

Kent, George Cantine, Jr., B.A. (Maryville), M.A., Ph.D. (Vanderbilt), Assistant Professor of Zoology, *Department of Zoology, Louisiana State University, University Station, Baton Rouge, La.*

Kepner, William Allison, A.B., A.M. (Franklin and Marshall), Ph.D. (Virginia), Sc.D. (Franklin and Marshall), Professor of Biology, University of Virginia, *29 University Place, University, Va.*

Kidder, George Wallace, A.B. (Oregon), M.A. (California), Ph.D. (Columbia), Assistant Professor of Biology, *Brown University, Providence, R. I.*

Kille, Frank Ralph, B.S. (College of Wooster), S.M., Ph.D. (Chicago), Professor of Zoology, and Dean of Men, *Carleton College, Northfield, Minn.*

Kimball, Richard F., A.B., Ph.D. (Johns Hopkins), Associate in Zoology, *Department of Biology, The Johns Hopkins University, Homewood, Baltimore 18, Md.*

Kindred, James Ernest, A.B. (Tufts), A.M., Ph.D. (Illinois), Professor of Anatomy, Medical School, *University of Virginia, Box 1873, University Station, Charlottesville, Va.*

King, Helen Dean, A.B. (Vassar), A.M., Ph.D. (Bryn Mawr), Member of The Wistar Institute of Anatomy and Biology, *The Wistar Institute, 36th St. and Woodland Ave., Philadelphia 4, Pa.*

King, Robert Leslie, A.B., Ph.D. (Pennsylvania), Professor of Zoology, *State University of Iowa, Iowa City, Iowa.*

Kinsey, Alfred Charles, B.S. (Bowdoin), Sc.D. (Harvard), Professor of Zoology, *Indiana University, Bloomington, Ind.*

Kirby, Harold, B.S. (Emory), M.A., Ph.D. (California), Professor of Zoology, *University of California, Berkeley, Calif.*

Kirkham, William Barri, B.A., M.A., Ph.D. (Yale), Consulting Director, Museum of Natural History, *220 State St., Springfield 5, Mass.*

Kleinholz, Lewis Hermann, B.S. (Colby), M.A., Ph.D. (Harvard), Fellow of the John Simon Guggenheim Memorial Foundation, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Kofoid, Charles Atwood, A.B., Sc.D. (Hon.) (Oberlin), A.M., Ph.D. (Harvard), D.Sc. (Hon.) (Wales), LL.D. (Hon.) (California), Professor of Zoology, Emeritus, *University of California, Berkeley, Calif.*

Kollros, Jerry John, S.B., Ph.D. (Chicago), Associate in Zoology, *Department of Zoology, State University of Iowa, Iowa City, Iowa.*

Kopac, M(ilan) J(ames), B.S., M.S. (Nebraska), Ph.D. (California), Visiting Assistant Professor of Biology, *Washington Square College, New York University, New York, N. Y.*

Koppányi, Theodore, Ph.D. (Vienna), Professor of Pharmacology, Georgetown University, School of Medicine, *3900 Reservoir Road, Washington, D. C.*

Kornhauser, Sidney Isaac, A.B. (Pittsburgh), A.M., Ph.D. (Harvard), Professor of Anatomy and Executive Secretary, School of Medicine, University of Louisville, *101 W. Chestnut St., Louisville, Ky.*

Krecker, Frederick Hartzler, A.B., Ph.D. (Princeton), A.M. (Cornell), Professor of Zoology, *Ohio University, Athens, Ohio.*

Kroc, Robert Louis, B.A., M.A. (Oberlin), Ph.D. (Wisconsin), Assistant Director of Research, Research Division, *The Maltine Company, 436 18th Street, Brooklyn 15, N. Y.*

Kropp, Benjamin, S.B., A.M., Ph.D. (Harvard), Assistant Professor of Embryology, *Queen's University Medical School, Kingston, Ontario, Canada.*

Kudo, R. R., D.Sc. (Tokio Imperial University), Professor of Zoology, Department of Zoology and Physiology, *322 Natural History Building, University of Illinois, Urbana, Ill.*

Kunkel, Beverly Waugh, Ph.B., Ph.D. (Yale), Professor of Biology, *Lafayette College, Easton, Pa.*

Kuntz, Albert, B.A. (Morningside), Ph.D. (Iowa), M.D. (St. Louis), Professor of Microanatomy, *St. Louis University School of Medicine, 1402 S. Grand Blvd., St. Louis, Mo.*

Lambert, William Vincent, B.S. (Nebraska), M.S. (Kansas State), Ph.D. (California), Associate Director, *Agricultural Experiment Station, Purdue University, Lafayette, Ind.*

LAMOREUX, WELFORD FORREST, B.S. (Iowa State), M.S., Ph.D. (Cornell), Geneticist and Director of Research, *Kimber Poultry Breeding Farm, Niles, Calif.*

LANDAUER, WALTER, Ph.D.Nat. (Heidelberg), Professor of Genetics, *Storrs Agricultural Experiment Station, Storrs, Conn.*

LANGLOIS, THOMAS HUXLEY, B.S., M.S. (Michigan), Ph.D. (Ohio State), Professor of Zoology and Director, Franz Theodore Stone Laboratory, Ohio State University, *Put-in-Bay, Ohio.*

LA RUE, GEORGE R., B.S. (Doane), A.M. (Nebraska), Ph.D. (Illinois), Professor of Zoology, *University of Michigan, Ann Arbor, Mich.*

LASHLEY, KARL SPENCER, A.B. (West Virginia), M.S. (Pittsburgh), Ph.D. (Johns Hopkins), Sc.D. (Pittsburgh, Chicago), Research Professor of Neuropsychology, Harvard University; Director, Yerkes Laboratories of Primate Biology, *Orange Park, Fla.*

LATIMER, HOMER BARKER, A.B., A.M., Ph.D. (Minnesota), Professor of Anatomy, *University of Kansas, Lawrence, Kan.*

LAWSON, CHESTER ALVIN, B.S. (Thiel), M.S., Ph.D. (Michigan), Michigan State College, *East Lansing, Mich.*

LEATHEM, JAMES HAIN, B.S. (Lebanon Valley), M.A., Ph.D. (Princeton), Assistant Professor of Zoology, *Department of Zoology, Rutgers University, New Brunswick, N. J.*

LEONARD, SAMUEL L(EESON), B.S. (Rutgers), M.S., Ph.D. (Wisconsin), Associate Professor of Zoology, Cornell University, *Ithaca, N. Y.*

LERNER, I. MICHAEL, B.S.A., M.S.A. (British Columbia), Ph.D. (California), Assistant Professor of Poultry Husbandry, *Division of Poultry Husbandry, University of California, Berkeley, Calif.*

LEVINE, PINCUS PHILIP, B.S. (College of the City of New York), D.V.M. (New York State Veterinary College), M.S., Ph.D. (Cornell), Professor of Poultry Diseases, *New York State Veterinary College, Ithaca, N. Y.*

LEWIS, FREDERIC THOMAS, A.M., M.D. (Harvard), James Stillman Professor of Comparative Anatomy, Emeritus, *Harvard Medical School, Boston, Mass.*

LIEBMANN, EMIL, Ph.D. (Vienna), Research Fellow in Biology, *Department of Biology, Princeton University, Princeton, N. J.*

LIGHT, SOL FELTY, A.B. (Park), M.A. (Philippines), M.S. (Princeton), Ph.D. (California), Professor of Zoology, *University of California, 4047 Life Sciences Building, Berkeley, Calif.*

LILLIE, FRANK RATTRAY, B.A., Sc.D. (Toronto), Ph.D. (Chicago), Sc.D. (Yale, Harvard), LL.D. (Johns Hopkins), Andrew MacLeish Distinguished Service Professor of Embryology, Emeritus, The University of Chicago, *5801 Kenwood Ave., Chicago 37, Ill.*

LILLIE, RALPH STAYNER, A.B., Sc.D. (Toronto), Ph.D. (Chicago), Professor of General Physiology, Emeritus, *The University of Chicago, Chicago 37, Ill.*

LINDEMAN, VERLUS FRANK, B.A. (North Central), M.S., Ph.D. (Iowa), Associate Professor of Zoology, Syracuse University, *12 Lyman Hall, Syracuse University, Syracuse, N. Y.*

LINDSEY, ARTHUR WARD, A.B. (Morningside), M.S., Ph.D. (State University of Iowa), Professor of Zoology, Denison University, *P. O. Box 612, Granville, Ohio.*

Lineburg, Bruce, A.B. (Ohio), A.M., Ph.D. (Johns Hopkins), Professor of Biology, *Lake Forest College, Lake Forest, Ill.*

Linsdale, Jean Myron, A.B., M.A. (Kansas), Ph.D. (California), Research Associate, Museum of Vertebrate Zoology, University of California, *Jamesburg Route, Monterey, Calif.*

Little, Clarence Cook, A.B., S.M., Sc.D. (Harvard), LL.D. (New Hampshire, Albion, New Mexico, Colby), Litt.D. (Maine), Director, *Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.*

Litwiller, Raymond, B.A. (Goshen College), Ph.D. (Chicago), Wilson Junior College, *6748 Constance Ave., Chicago 49, Ill.*

Loefer, John B., B.A. (Lawrence), M.S., Ph.D. (New York), Associate Professor of Biology and Chemistry, *Berea College, Berea, Ky.*

Long, Joseph Abraham, S.B., A.M., Ph.D. (Harvard), Professor of Embryology, *3075 Life Sciences Building, University of California, Berkeley, Calif.*

Loosanoff, Victor L., B.S. (Washington), Ph.D. (Yale), Director, *U. S. Fish and Wildlife Service Laboratory, Milford, Conn.*

Lucas, Alfred Martin, A.B. (Wabash), Ph.D. (Washington University), Cytopathologist, U. S. Regional Poultry Research Laboratory, *Box 270, East Lansing, Mich.*

Lucas, Miriam Scott (Mrs. A. M.), B.S., Ph.D. (Pennsylvania), Instructor in Biological Sciences, *Basic College, Michigan State College, East Lansing, Mich.*

Luce, Wilbur Marshall, B.S. (Bates), M.S., Ph.D. (Illinois), Assistant Professor of Zoology, University of Illinois, *345 Natural History Building, Urbana, Ill.*

Ludwig, Daniel, A.B. (Ursinus), Ph.D. (Pennsylvania), Associate Professor of Biology, *New York University, University Heights, N. Y.*

Lund, Elmer Julius, Ph.D. (Johns Hopkins), Professor of Physiology and Biophysics, *University of Texas, Austin, Texas.*

Lund, Everett Eugene, B.S. (Iowa State College), M.A., Ph.D. (California), Associate Professor of Biology, *Department of Biology, Alfred University, Alfred, N. Y.*

Lutz, Brenton Reid, B.S., A.M., Ph.D. (Boston), Professor of Biology, *Boston University, 688 Boylston St., Boston, Mass.*

Luyet, Basile Joseph, A.B. (Fribourg), L.Sc., D.Sc. (Biology), D.Sc. (Physics), (Geneva), Professor of Biology, *St. Louis University Medical School, St. Louis, Mo.*

Lynch, Clara J., A.B. (Smith), A.M., Ph.D. (Columbia), Associate, Rockefeller Institute, *66th St. and York Ave., New York, N. Y.*

Lynch, James Eric, A.B., A.M. (Nebraska), Ph.D. (California), Professor, *School of Fisheries, University of Washington, Seattle, Wash.*

Lynch, Ruth Stocking (Mrs. Vernon Lynch), A.B. (Goucher), Ph.D. (Johns Hopkins), Assistant to Dr. H. S. Jennings, *Department of Zoology, University of California, Los Angeles 24, Calif.*

Lynn, William Gardner, A.B., Ph.D. (Johns Hopkins), Associate Professor of Biology, *Department of Biology, The Catholic University of America, Washington, D. C.*

MacArthur, John Wood, A.B. (Oberlin), M.A. (Wabash), Ph.D. (Chicago), F.R.S.C., Associate Professor of Genetics, University of Toronto, *Department of Biology, University of Toronto, Toronto, Ontario, Canada.*

MacDougall, Mary Stuart, A.B. (Randolph-Macon), M.S. (Chicago), Ph.D. (Columbia), Sc.D. (Montpellier), Professor of Zoology and Chairman of the Department of Biology, *Agnes Scott College, Decatur, Ga.*

MacDowell, Edwin Carleton, A.B. (Swarthmore), S.M., Sc.D. (Harvard), Resident Investigator, Station of Experimental Evolution, *Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y.*

McCay, Clive Maine, A.B. (Illinois), M.S. (Iowa State), Ph.D. (California), *Nutrition Laboratory, Dairy Building, Cornell University, Ithaca, N. Y.*

McClure, Charles F. W., A.B., A.M. (Princeton), D.Sc. (Hon.) (Columbia), Professor of Comparative Anatomy, Emeritus, *Princeton University, Princeton, N. J.*

McCulloch, Irene, A.B., A.M. (Kansas), Ph.D. (California), Professor of Zoology, Allan Hancock Foundation, University of Southern California, *Los Angeles, Calif.*

McCutcheon, Frederick Harold, Ph.D. (Duke), Professor of Zoology, *North Carolina State College, Raleigh, N. C.*

McEwen, Robert Stanley, A.B., A.M. (Western Reserve), Ph.D. (Columbia), Professor of Zoology, *Oberlin College, Oberlin, Ohio.*

McGregor, James Howard, B.S. (Ohio State), M.A., Ph.D. (Columbia), Professor of Zoology, Emeritus, *Columbia University, New York, N. Y.*

McIndoo, Norman Eugene, A.B., A.M. (Indiana), Ph.D. (Pennsylvania), Senior Entomologist, Emeritus, Bureau of Entomology, U. S. Department of Agriculture, *7225 Blair Road, Takoma Park, Washington 12, D. C.*

Magath, Thomas Byrd, Ph.B. (Emory), M.S. (Millikin), Ph.D., M.D. (Illinois), Professor of Pathology and Parasitology, University of Minnesota, and Consulting Physician, *Mayo Clinic, Rochester, Minn.*

Maluf, N(oble) S(uydam) Rustum, B.Sc. (American University, Cairo), M.S., Ph.D. (Cornell), Medical Student, *Vanderbilt Hall, Harvard Medical School, Boston, Mass.*

Manter, Harold Winfred, A.B. (Bates), A.M., Ph.D. (Illinois), Professor of Zoology, University of Nebraska, *204 Bessey Hall, University of Nebraska, Lincoln, Nebr.*

Manwell, Reginald Dickinson, A.B., A.M. (Amherst), D.Sc. (Johns Hopkins), Professor of Zoology, Syracuse University, *25 Lyman Hall, Syracuse University, Syracuse, N. Y.* (On leave for military service, '43, Capt. Sanitary Corps. Chief of Section of Protozoology, *Army Medical School, Army Medical Center, Washington, D. C.*)

†**Mark, Edward L.**, A.B. (Michigan), Ph.D. (Leipzig), LL.D. (Michigan, Wisconsin), Hersey Professor of Anatomy, Emeritus, Harvard University, *109 Irving St., Cambridge, Mass.*

Marsh, Gordon, A.B., A.M., Ph.D. (Texas), Assistant Professor of Zoology, *Department of Zoology, State University of Iowa, Iowa City, Iowa.*

Marshall, Ruth, B.S., M.S. (Wisconsin), Ph.D. (Nebraska), Professor of Zoology, Emeritus, Rockford College, *Wisconsin Dells, Wis.*

Marshall, William Stanley, B.S. (Swarthmore), Ph.D. (Leipzig), Professor of Entomology, Emeritus, University of Wisconsin, *139 E. Gilman St., Madison, Wis.*

Marsland, Douglas A., B.S. (New York), M.A. (Columbia), Ph.D. (New York), Associate Professor of Biology, *Department of Biology, New York University, Washington Square, New York, N. Y.*

Martin, Walter Edwin, B.Educ. (North Illinois State Teachers), M.S., Ph.D. (Purdue), Associate Professor of Zoology, DePaul University, *R.R. No. 4, Greencastle, Ind.*

Mast, Samuel Ottmar, B.S. (Michigan), Ph.D. (Harvard), M.Pd. (Michigan Normal), Sc.D. (Michigan), Professor of Zoology, Emeritus, *The Johns Hopkins University, Baltimore, Md.*

Matthews, Samuel A., B.S., M.A. (Boston), Ph.D. (Harvard), Professor of Biology, *Williams College, Williamstown, Mass.*

Mattox, Norman Thomas, A.B. (Miami), M.S., Ph.D. (Illinois), *Department of Zoology, Miami University, Oxford, Ohio.*

Mavor, James Watt, B.A. (Cambridge), M.A., Ph.D. (Harvard), Professor of Biology and Head of the Department, *Union College, Schenectady, N. Y.*

May, Raoul Michel, A.B. (Stanford), A.M., Ph.D. (Harvard), Docteur ès Sciences, M.D. (Paris); Chef de Travaux, *Laboratoire d'Anatomie et d'Histologie Comparées, Sorbonne, Paris 5^e, France.*

Mayr, Ernst, Ph.D. (Berlin), Curator, Whitney-Rothschild Collection, *American Museum of Natural History, New York 24, N. Y.*

Mazia, Daniel, A.B., Ph.D. (Pennsylvania), Associate Professor of Zoology, *Department of Zoology, University of Missouri, Columbia, Mo.* (On leave for military service, '42. 1st Lt., U. S. A.)

Mead, Albert Davis, A.B. (Middlebury), A.M. (Brown), Ph.D. (Chicago), Sc.D. (Pittsburgh, Middlebury, Rhode Island State College), LL.D. (Brown), Professor of Biology, Emeritus, Brown University, *283 Wayland Ave., Providence, R. I.*

Melampy, Robert M., B.S. (Wilmington College), M.A. (Haverford), Ph.D. (Cornell), Associate Professor of Zoology, *Louisiana State University, Baton Rouge, La.*

Melander, A(xel) L(eonard), B.S., M.S. (Texas), Sc.D. (Harvard), Professor of Biology, Emeritus, College of the City of New York, New York, N. Y., Research Associate, University of California, *4670 Rubidoux Lane, Riverside, Calif.*

Mendoza, Guillermo, B.S., M.S., Ph.D. (Northwestern), Associate Professor of Zoology, *Department of Zoology, Grinnell College, Grinnell, Iowa.*

Merriman, Daniel, B.S., M.S. (Washington), Ph.D. (Yale), Assistant Professor of Biology, Director of the Bingham Oceanographic Laboratory, *Yale University, New Haven, Conn.*

Metz, Charles Baker, A.B. (Johns Hopkins), Ph.D. (California Institute of Technology), Instructor in Biology, Wesleyan University. (On leave, National Research Council Fellow, *Department of Zoology, Indiana University, Bloomington, Ind.*)

Metz, Charles William, B.A. (Pomona), Ph.D. (Columbia), Professor of Zoology, *Zoological Laboratory, University of Pennsylvania, Philadelphia 4, Pa.*

Meyer, A(rthur) W(illiam), B.S. (Wisconsin), M.D. (Johns Hopkins), Professor of Anatomy, Emeritus, Stanford University, *121 Waverly St., Palo Alto, Calif.*

Meyer, Roland Kenneth, A.B. (Milton), A.M., Ph.D. (Wisconsin), Professor of Zoology, *Department of Zoology, University of Wisconsin, Madison, Wis.*

Mickey, George H(enry), A.B. (Baylor), M.S. (Oklahoma), Ph.D. (Texas), Associate Professor of Zoology, *Department of Zoology, Louisiana State University, Baton Rouge 3, La.*

Middleton, Austin Ralph, A.B., Ph.D. (Johns Hopkins), Professor of Zoology, Director of the Department of Biology, *University of Louisville, 1st and Chestnut Sts., Louisville, Ky.*

Miller, Alden Holmes, A.B., M.A., Ph.D. (California), Associate Professor of Zoology and Director, *Museum of Vertebrate Zoology, University of California, Berkeley, Calif.*

Miller, James Albert, A.B. (Wooster), Ph.D. (Chicago), Assistant Professor of Histology and Embryology, *Division of Anatomy, College of Medicine, University of Tennessee, Memphis 3, Tenn.*

Miller, John, A(llen), B.A. (Ashland), M.S., Ph.D. (Ohio State), Associate Professor of Zoology, *Ohio State University, Columbus, Ohio.*

Miller, Robert Cunningham, A.B. (Greenville), M.A., Ph.D. (California), Director, *California Academy of Sciences, San Francisco, Calif.*

Milne, Lorus J(ohnson), B.A. (Toronto), M.A., Ph.D. (Harvard), Research Associate in War Visual Problems, *Johnson Foundation, University of Pennsylvania, Philadelphia, Pa.*

Minnich, Dwight Elmer, A.B. (Miami), Ph.D. (Harvard), Professor of Zoology and Chairman of the Department, *University of Minnesota, Minneapolis, Minn.*

Mizelle, John Dary, B.S., M.S. (Louisiana), Ph.D. (Illinois), Associate Professor of Biology, *Department of Biology, University of Notre Dame, Notre Dame, Ind.*

Moody, Paul Amos, A.B. (Morningside), Ph.D. (Michigan), Professor of Zoology and Director of Graduate Study, *Department of Zoology, University of Vermont, Burlington, Vt.*

Moore, Carl R., B.S., A.M. (Drury), Ph.D. (Chicago), Professor of Zoology and Chairman of the Department, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Moore, John Alexander, A.B., M.A., Ph.D. (Columbia), Assistant Professor of Zoology, *Barnard College, Columbia University, New York 27, N. Y.*

* Moore, J. Percy, Ph.D. (Pennsylvania), Professor of Zoology, Emeritus, *University of Pennsylvania, Philadelphia, Pa.*

Morgan, Ann Haven, A.B., Ph.D. (Cornell), Professor of Zoology, *Mount Holyoke College, South Hadley, Mass.*

Morgan, Banner Bill, B.S., M.S. (Oregon State), M.A. (Iowa State), Ph.D. (Wisconsin), Associate Professor and Parasitologist in Veterinary Science, *Department of Veterinary Science, College of Agriculture, University of Wisconsin, Madison, Wis.*

Morgan, William Pitt, A.B. (Indiana Central), A.M., Ph.D. (Indiana), Professor of Biology, Indiana Central College, *Route 6, Box 414, Indianapolis, Ind.*

Morrill, Charles V., A.M., Ph.D. (Columbia), Associate Professor of Anatomy, Cornell University Medical College, *1300 York Ave., New York, N. Y.*

Mosher, Edna, B.S. (Cornell), Ph.D. (Illinois), Professor of Biology, Emeritus, Adelphi College, Garden City, N. Y. *R. R. no. 1, Newport, Nova Scotia, Canada.*

Mossman, Harland Winifred, B.S. (Allegheny), M.S., Ph.D. (Wisconsin), Associate Professor of Anatomy, *University of Wisconsin, Madison, Wis.*

Muller, Hermann, J., A.B., A.M., Ph.D. (Columbia), D.Sc. (Edinburgh), Professor of Zoology, *Indiana University, Bloomington, Ind.*

Murray, Margaret Ransone, A.B. (Goucher), M.S. (Washington), Ph.D. (Chicago), Associate in Surgery, *College of Physicians and Surgeons, Columbia University, 630 W. 168th St., New York, N. Y.*

Myers, Earl Hamlet, B.S. (Washington State College), Ph.D. (California), *308 Oceanographic Laboratory, Woods Hole, Mass.*

Myers, George Sprague, A.B., A.M., Ph.D. (Stanford), Professor of Biology, *Stanford University, Palo Alto, Calif.*

Nabours, Robert K., Ph.D. (Chicago), Professor of Zoology and Experiment Station Zoologist, *Kansas State College, Manhattan, Kan.*

Nabrit, Samuel Milton, B.S. (Morehouse), M.S., Ph.D. (Brown), Professor of Biology, *Atlanta University, Atlanta, Ga.*

Nalbandov, Andrew Vladimir, B.S. (Munich), M.S. (Oklahoma), Ph.D. (Wisconsin), Assistant Professor of Animal Physiology, *University of Illinois, Urbana, Ill.*

Nelsen, Olin Everett, A.M. (Brown), Ph.D. (Pennsylvania), Assistant Professor of Zoology, *University of Pennsylvania, Philadelphia 4, Pa.*

Nelson, Thurlow C(hristian), B.A., D.Sc. (Rutgers), Ph.D. (Wisconsin), Professor of Zoology, *Rutgers University;* Biologist, State Board of Shell Fisheries, *New Brunswick, N. J.*

Nelson, Warren Otto, A.B. (Augustana), Sc.M. (Iowa), Ph.D. (New York), Professor of Anatomy, Department of Anatomy, *State University of Iowa, College of Medicine, Iowa City, Iowa.*

Newman, Horatio Hackett, B.A., Sc.D. (McMaster), Ph.D. (Chicago), Professor of Zoology, Emeritus, The University of Chicago, *173 Devon Drive, Clearwater, Fla.*

Nicholas, John Spangler, S.B., S.M. (Gettysburg), Ph.D. (Yale), Sterling Professor of Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

Nigrelli, Ross F., B.S. (Pennsylvania State College), M.S., Ph.D. (New York), Assistant Director and Pathologist, New York Aquarium; Consultant U. S. Fish and Wildlife Service; Consultant Bingham Oceanographic Laboratory; Yale University, *New York Aquarium, New York Zoological Society, Bronx, New York, N. Y.*

Noland, Lowell Evan, B.A. (De Pauw), M.A., Ph.D. (Wisconsin), Professor of Zoology, *Biology Building, University of Wisconsin, Madison, Wis.*

Novikoff, Alex Benjamin, B.S., M.A., Ph.D. (Columbia), Instructor in Biology, *Department of Biology, Brooklyn College, Ave. H and Bedford Ave., Brooklyn, N. Y.*

Nuttycombe, John William, B.S. (Virginia Polytechnic Institute), Ph.D. (Virginia), Professor of Zoology, *LeConte Hall, University of Georgia, Athens, Ga.*

OBRESKOVE, VASIL, B.S., M.S. (Syracuse), Ph.D. (Harvard), Professor of Biology, *Bard College, Annandale-on-Hudson, N. Y.*

OKKELBERG, PETER, A.B., A.M. (Minnesota), Ph.D. (Michigan), Professor of Zoology and Assistant Dean of the Horace H. Rackham School of Graduate Studies, University of Michigan, *1220 Fair Oaks, Ann Arbor, Mich.*

OLD, MARCUS CALVIN, B.A., M.A. (Lehigh), Ph.D. (Michigan), Professor and Chairman of the Department of Biology, Hofstra College, *125 Duncan Rd., Hempstead, N. Y.*

OLIVER, CLARENCE PAUL, A.B., M.A., Ph.D. (Texas), Professor of Zoology and Director Dight Institute, *Department of Zoology, University of Minnesota, Minneapolis, Minn.*

OPPENHEIMER, JANE M., B.A. (Bryn Mawr), Ph.D. (Yale), Assistant Professor of Biology, *Bryn Mawr College, Bryn Mawr, Pa.*

O'ROKE, EARL CLEVELAND, A.B., M.A. (Kansas), Ph.D. (California), Associate Professor of Forest Zoology, *4052 Natural Science Building, University of Michigan, Ann Arbor, Mich.*

ORTENBURGER, ARTHUR IRVING, A.B., M.S., Ph.D. (Michigan), Professor of Zoology and Chairman of the Department, University of Oklahoma; Curator of Vertebrates, Museum of Zoology; Vertebrate Zoologist, Oklahoma Biological Survey, *University of Oklahoma, Norman, Okla.*

OSBORN, CLINTON MORRIS, A.B. (Bates), M.A., Ph.D. (Harvard), Assistant Professor of Histology and Embryology, *Department of Anatomy, Ohio State University, Columbus, Ohio.* (On leave for military service, '42, U. S. A.)

OSBURN, RAYMOND C., Ph.D. (Columbus), Professor of Zoology, Emeritus, *Ohio State University, Columbus, Ohio.*

PACE, DONALD METCALF, A.B. (Susquehanna), M.A., Ph.D. (Duke), Associate Professor of Physiology, *Department of Physiology and Pharmacology, University of Nebraska, Lincoln, Nebr.*

PACKARD, CHARLES, B.S., M.S. (Syracuse), Ph.D. (Columbia), Sc.D. (Hon.) (Syracuse), Resident Director, Marine Biological Laboratory, *Woods Hole, Mass.*

PAINTER, THEOPHILUS SCHICKEL, A.B. (Roanoke), A.M., Ph.D., Sc.D. (Yale), Professor of Zoology, *University of Texas, Austin 12, Texas.*

PARK, ORLANDO, B.S., Ph.D. (Chicago), Professor of Zoology, *Department of Zoology, Northwestern University, Evanston, Ill.*

PARK, THOMAS, S.B., Ph.D. (Chicago), Associate Professor of Zoology, Associate Dean, Division of Biological Sciences, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

†PARKER, GEORGE HOWARD, S.B., D.Sc. (Harvard), Sc.D. (Colby), Professor of Zoology, Emeritus, Biological Laboratories, Harvard University, *16 Berkeley St., Cambridge 38, Mass.*

PARMENTER, CHARLES LEROY, A.B., M.A. (Southern California), Ph.D. (Pennsylvania), Professor of Zoology, *Zoological Laboratory, University of Pennsylvania, Philadelphia 4, Pa.*

PARPART, ARTHUR KEMBLE, B.A., M.A. (Amherst), Ph.D. (Pennsylvania), Associate Professor of Physiology, *Princeton University, Princeton, N. J.*

PARSHLEY, H(OWARD) M(ADISON), A.B., A.M., Sc.D. (Harvard), Professor of Zoology, *Smith College, Northampton, Mass.*

PATTEN, BRADLEY MERRILL, A.B. (Dartmouth), A.M., Ph.D. (Harvard), Professor of Anatomy and Director of Anatomical Laboratories, University of Michigan, Medical School, *2500 East Medical Bldg., Ann Arbor, Mich.*

PATTERSON, JOHN THOMAS, B.S., Sc.D. (Wooster), Ph.D. (Chicago), Professor of Zoology; Director of Zoological Research, University of Texas, *University Station, Austin 12, Texas.*

PATTERSON, THOMAS LEON, A.B. (Clark), A.M. (Kansas City), M.S., Ph.D. (Chicago), Professor of Physiology, University of Detroit, College of Dentistry, also Research Professor of Physiology, Wayne University, College of Medicine, *1512 St. Antoine St., Detroit, Mich.*

PAYNE, FERNANDUS, A.B., A.M. (Indiana), Ph.D. (Columbia), Professor of Zoology, Dean of the Graduate School, Indiana University, *620 Ballentine Rd., Bloomington, Ind.*

PAYNE, NELLIE M., B.S., M.S. (Kansas State), Ph.D. (Minnesota), Research Entomologist, *American Cyanamid Company, Boston Post Road, Stamford, Conn.*

PEARSE, ARTHUR SPERRY, B.S., A.M., LL.D. (Nebraska), Ph.D. (Harvard), Professor of Zoology, *Duke University, Durham, N. C.*

PEARSON, NATHAN EVERETT, A.B., A.M., Ph.D. (Indiana), Professor of Zoology, *Butler University, Indianapolis, Ind.*

PENNAK, ROBERT WILLIAM, B.S., M.S., Ph.D. (Wisconsin), Associate Professor of Biology, *Biology Department, University of Colorado, Boulder, Colo.*

PENNEY, JAMES THEOPHILUS, A.B., M.A., Ph.D. (North Carolina), Professor of Biology, *University of South Carolina, Columbus, S. C.*

PERKINS, HENRY F., A.B. (Vermont), Ph.D. (Johns Hopkins), Professor of Zoology and Director of Fleming Museum, University of Vermont, *205 S. Prospect St. Burlington, Vt.*

PETRUNKEVITCH, ALEXANDER, Ph.D. (Freiburg), M.A. (Hon.) (Yale), D.Sc. (Hon.) (Puerto Rico), Professor of Zoology, Emeritus, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

PFEIFFER, CARROLL A., B.A. (Marietta), M.S., Ph.D. (Iowa), Research Assistant (Assistant Professor) in Anatomy, *Yale University, Department of Anatomy, 333 Cedar St., New Haven, Conn.*

PFEIFFER, ISABELLE WEED, B.A. (Wisconsin), M.S., Ph.D. (Iowa), Honorary Fellow in Zoology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

PHELPS, AUSTIN, Ph.B., Ph.D. (Yale), Assistant Professor of Zoology, University of Texas, *Box 1972 University Station, Austin 12, Texas.*

PHILIPS, FREDERICK STANLEY, B.A. (Columbia), Ph.D. (Rochester), Research Assistant, *Department of Pharmacology and Toxicology, Yale University Medical School, New Haven, Conn.*

PHILPOTT, CHARLES HUGHES, A.B., A.M., Ph.D. (Missouri), Principal, *Harris Teachers College, St. Louis, Mo.*

PIATT, JEAN, B.S. (Butler), M.A. (Haverford), Ph.D. (Yale), Assistant Professor of Anatomy, *University of Pennsylvania, Philadelphia 4, Pa.*

PICKFORD, GRACE EVELYN, Nat. Sci. Tripos (Cambridge), Ph.D. (Yale), Research Assistant, Bingnam Oceanographic Laboratory, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

PIKE, FRANK HENRY, A.B. (Indiana), Ph.D. (Chicago), Associate Professor of Physiology, Emeritus, Special Lecturer in Physiology, Columbia University, *437 West 59th St., New York 19, N. Y.*

PINCUS, GREGORY, B.S. (Cornell), M.S., D.Sc. (Harvard), Visiting Professor of Experimental Zoology, *Clark University, Worcester, Mass.*

PINNEY, MARY EDITH, A.B., A.M. (Kansas), Ph.D. (Bryn Mawr), Professor of Zoology, *Milwaukee-Downer College, Milwaukee, Wis.*

PLAGGE, JAMES C. S.B., Ph.D. (Chicago), Assistant Professor of Anatomy, *University of Illinois College of Medicine, 1853 W. Polk St., Chicago 12, Ill.*

PLOUGH, HAROLD HENRY, A.B. (Amherst), M.A., Ph.D. (Columbia), Professor of Biology, Amherst College, *53 Dana St., Amherst, Mass.* (On leave for military service, '42. Capt., U. S. A.)

POLLISTER, ARTHUR WAGG, A.B. (Bates), A.M., Ph.D. (Columbia), Associate Professor of Zoology, *Columbia University, New York, N. Y.*

POMERAT, CHARLES MARC, A.B. (Clark), A.M., Ph.D. (Harvard), Professor of Anatomy, *Medical branch University of Texas, Galveston, Texas.*

PORTER, DALE ALBERT, A.B. (Kalamazoo College), M.S. (Kansas State College), Sc.D. (Johns Hopkins), Parasitologist, U. S. Regional Animal Disease Research Laboratory, *P.O. Drawer 952, Auburn, Ala.*

POTTER, GEORGE EDWIN, B.S. (Ottawa University, Kansas), M.S., Ph.D. (Iowa), Professor of Biology, Texas Agricultural and Mechanical College, *Box 1, F. E., College Station, Texas.*

POULSON, DONALD FREDERICK, B.S., Ph.D. (California Institute of Technology), Assistant Professor of Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

POWER, MAXWELL ELLIOT, A.B. (Indiana), M.S. (Oklahoma), Ph.D. (Yale), Instructor in Biology, *Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

POWERS, EDWIN B., A.B. (Trinity), M.S. (Chicago), Ph.D. (Illinois), Professor of Zoology, Head of the Departments of Zoology and Entomology, *University of Tennessee, Knoxville, Tenn.*

PRATT, HENRY SHERRING, A.B. (Michigan), A.M., Ph.D. (Leipzig), Professor of Biology, Emeritus, *Haverford College, Haverford, Pa.*

PRICE, DOROTHY, B.S., Ph.D. (Chicago), Research Associate in Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

PRICE, JOHN WORTHINGTON, B.S., M.S., Ph.D. (Ohio State), Professor of Zoology, *Ohio State University, Columbus, Ohio.*

PROSSER, C(LIFFORD) LADD, A.B. (Rochester), Ph.D. (Johns Hopkins), Assistant Professor of Zoology, *University of Illinois, Urbana, Ill.*

PRYTHERCH, HERBERT FRANCIS, B.S. (Syracuse), Ph.D. (Pennsylvania), Director, *U. S. Fishery Biological Laboratory, Beaufort, N. C.*

PUCKETT, WILLIAM OLIN, A.B. (Davidson), A.M. (North Carolina), Ph.D. (Princeton), Assistant Professor of Comparative Anatomy and Vertebrate Embryology, *Department of Biology, Princeton University, Princeton, N. J.*

RAHN, HERMANN, A.B. (Cornell), Ph.D. (Rochester), Instructor in Physiology, *Department of Physiology, Rochester University, Rochester, N. Y.*

Ramsey, Robert Weberg, B.S., M.S., Ph.D. (New York), Associate Professor of Physiology, *Department of Physiology and Pharmacology, Medical College of Virginia, Richmond, Va.*

Rand, Herbert Wilbur, A.B. (Allegheny, Harvard), A.M., Ph.D. (Harvard), Associate Professor of Zoology, Emeritus, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Randall, Walter Clark, B.A. (Taylor), M.S., Ph.D. (Purdue), Instructor in Physiology, *St. Louis University, School of Medicine, St. Louis, Mo.*

Rankin, John Stewart, Jr., A.B., M.A. (Wesleyan), Ph.D. (Duke), Instructor in Zoology, *Department of Zoology, University of Connecticut, Storrs, Conn.*

Rawles, Mary Elbert, A.B. (Randolph-Macon Woman's College), S.M., Ph.D. (Chicago), Research Associate in Embryology, *Department of Biology, The Johns Hopkins University, Baltimore, Md.*

Redfield, Alfred Clarence, B.S., Ph.D. (Harvard), Professor of Physiology, Harvard University, (on leave), Associate Director Woods Hole, *Oceanographic Institution, Woods Hole, Mass.*

Reese, Albert Moore, A.B., Ph.D. (Johns Hopkins), Professor of Zoology, *West Virginia University, Morgantown, W. Va.*

Rehn, James Abram Garfield, Curator of Entomology, Corresponding Secretary, *Academy of Natural Sciences, 1900 Race Street, Philadelphia, Pa.*

Reid, (Willard) Malcolm, B.S. (Monmouth), M.S., Ph.D. (Kansas State), Assistant Professor of Biology and Chairman of the Department, *Monmouth College, Monmouth, Ill.*

Reinhard, Edward G., A.B., A.M. (Woodstock), Ph.D. (Minnesota), Professor of Biology and Head of the Department, *The Catholic University of America, Washington, D. C.*

Reynolds, Bruce Dodson, B.S. (Virginia), Sc.D. (Johns Hopkins), Professor of Biology, *University of Virginia, University, Va.*

Rhodes, Robert Clinton, A.B. (Henderson Brown), A.B., A.M. (Vanderbilt), Ph.D. (California), Professor and Chairman of the Department of Biology, *Emory University, Emory, Ga.*

Rice, Edward Loranus, A.B. (Wesleyan), Ph.D. (Munich), D.Sc. (Wesleyan), Professor of Zoology, Emeritus, *Ohio Wesleyan University, 265 N. Sandusky St., Delaware, Ohio.*

Rich, Willis H., B.S. (Lombard), A.B., A.M., Ph.D. (Stanford), Professor of Biology, *Stanford University, Calif.*

Richards, Albert Glenn, A.B. (Georgia), Ph.D. (Cornell), Associate Professor of Zoology and Entomology, *Department of Entomology, University Farm, University of Minnesota, St. Paul 8, Minn.*

Richards, Aute, B.A. (Kansas), M.A. (Wisconsin), Ph.D. (Princeton), Sc.D. (Marietta), Professor of Zoology, Director, School of Applied Biology, University of Oklahoma, *434 Chautauqua, Norman, Okla.*

Richards, Mildred Hoge, A.B. (Goucher), A.M., Ph.D. (Columbia), *434 Chautauqua, Norman, Okla.*

Richards, Oscar W., A.B., A.M. (Oregon), Ph.D. (Yale), Chief Biologist, *American Optical Company, Scientific Instrument Division, Buffalo 15, N. Y.*

Ricker, William Edwin, B.A., M.A., Ph.D. (Toronto), Associate Professor of Zoology, *Department of Zoology, Indiana University, Bloomington, Ind.*

Riddle, Oscar, A.B. (Indiana), Ph.D. (Chicago), LL.D. (Indiana), Research Staff, Carnegie Institution, Station for Experimental Evolution, *Cold Spring Harbor, Long Island, N. Y.*

Riley, William Albert, B.S. (DePauw), Ph.D. (Cornell), Professor of Zoology, Emeritus, *University of Minnesota, Minneapolis, Minn.*

Ringoen, Adolph R., B.A. (Iowa), M.A., Ph.D. (Minnesota), Professor of Zoology, *University of Minnesota, Minneapolis, Minn.*

Risley, Paul Lemuel, A.B. (Albion), M.S., Ph.D. (Michigan), Professor of Biology and Chairman of the Department, *Department of Biology, University of Oregon, Eugene, Ore.*

Roberts, Elmer, B.S., Ph.D. (Illinois), Professor of Animal Genetics, *University of Illinois, College of Agriculture, Urbana, Ill.*

Roberts, Howard Radclyffe, B.S. (Princeton), Ph.D. (Pennsylvania), Research Associate in Entomology, *Academy of Natural Sciences, 19th and Race Streets, Philadelphia, Pa.*

Rogers, Charles Gardner, A.B., A.M. (Syracuse), Ph.D. (California), Sc.D. (Syracuse), Professor of Comparative Physiology, Oberlin College, *378 Reamer Place, Oberlin, Ohio.*

Rogick, Mary Dora, A.B., A.M. (Nebraska), Ph.D. (Ohio State), Professor of Biology, College of New Rochelle, *25 Prospect St., Apt. 1-K, New Rochelle, N. Y.*

Romanoff, Alexis Lawrence, B.S., M.S., Ph.D. (Cornell), Associate Professor (Experimental Avian Embryology), *Department of Poultry Husbandry, Cornell University, Ithaca, N. Y.*

Romer, Alfred Sherwood, A.B. (Amherst), Ph.D. (Columbia), Professor of Zoology, Biological Laboratories, *Harvard University, Cambridge, Mass.*

Root, Raymond Willard, B.A. (Milton), A.M., Ph.D. (Duke), Assistant Professor of Biology, *College of the City of New York, 139th St. and Convent Ave., New York, N. Y.*

Rose, S(ylvan) Meryl, B.A., M.A. (Amherst), Ph.D. (Columbia), Assistant Professor of Zoology, *Department of Zoology, Smith College, Northampton, Mass.*

Rosenberg, Lauren Emery, B.S. (Knox), M.A., Ph.D. (California), *Naval Medical Research Unit no. 1, Life Sciences Building, University of California, Berkeley 4, Calif.*

Rosene, Hilda Florence, B.S., M.S. (Washington), Ph.D. (Texas), Associate Professor of Physiology, *University of Texas, Austin 12, Texas.*

Rudnick, Dorothea, Ph.B., Ph.D. (Chicago), Assistant Professor of Biology, *Albertus Magnus College, 700 Prospect St., New Haven, Conn.*

Rugh, Roberts, A.B., M.A. (Oberlin), Ph.D. (Columbia), Associate Professor of Zoology, *Washington Square College, New York University, New York, N. Y.*

Rulon, Olin, B.A. (Park), M.S. (Oklahoma), Ph.D. (Chicago), Assistant Professor of Biology, *Wayne University, Detroit, Mich.*

Russell, William Lawson, A.B. (Oxford), Ph.D. (Chicago), Research Associate, *Jackson Memorial Laboratory, Bar Harbor, Me.*

Ruth, Elbert Bresee, A.B. (Indiana), A.M., Ph.D. (Wisconsin), Assistant Professor of Anatomy, *Department of Anatomy, College of Medicine, Eden Ave., University of Cincinnati, Cincinnati, Ohio.*

RUTHVEN, ALEXANDER G., B.S. (Morningside), Ph.D. (Michigan), Sc.D. (Kalamazoo, Michigan College of Mining and Technology), LL.D. (Albion, Northwestern, Morningside, Denison, Tulane, California, New Mexico), President, *University of Michigan, Ann Arbor, Mich.*

RYAN, FRANCIS JOSEPH, A.B., M.A., Ph.D. (Columbia), Assistant Professor of Zoology, *Department of Zoology, Columbia University, New York, N. Y.*

SAMPSON, MYRA MELISSA, Ph.B. (Brown), A.M., Ph.D. (Michigan), Professor of Zoology, *Department of Zoology, Smith College, Northampton, Mass.*

SANDOW, ALEXANDER, B.S. (Massachusetts State), M.A. (Columbia), Ph.D. (New York), Assistant Professor of Biology, *Washington Square College of Arts and Science, New York University, New York, N. Y.*

SANDSTROM, CARL JOHAN, B.S., Ph.D. (Chicago), Associate Professor of Biology, *Department of Biology, New York University, University Heights, New York, N. Y.*

SAWIN, PAUL BALDWIN, B.S., (Cornell), M.S. (Kansas State), Sc.D. (Harvard), Assistant Professor of Biology, *Brown University, Providence, R. I.*

SAYLES, LEONARD P., A.B., A.M., Ph.D. (Brown), Associate Professor of Biology, *College of the City of New York, 139th and Convent Ave., New York, N. Y.*

SCHAEFFER, ASA ARTHUR, A.B. (Franklin and Marshall), Ph.D. (Johns Hopkins), Professor of Biology, *Temple University, Philadelphia, Pa.*

SCHARRER, BERTA (MRS. E. SCHARRER), Ph.D. (University of Munich), Instructor in Anatomy (Research), *Department of Anatomy, University of Colorado School of Medicine, Denver 7, Colo.*

SCHECHTER, VICTOR, B.S. (College of the City of New York), M.A., Ph.D. (Columbia), Assistant Professor of Biology, *College of the City of New York, New York, N. Y.*

SCHIEDT, RICHARD C. F., Ph.D. (Pennsylvania), Sc.D. (Hon.) (Franklin and Marshall), *1043 Wheatland Ave., Lancaster, Pa.*

SCHMIDT, IDA GENTHER, B.A. (Mount Holyoke), M.A. (Wisconsin), Ph.D. (Washington), Assistant Professor of Anatomy, *College of Medicine, Eden Ave., University of Cincinnati, Cincinnati, Ohio.*

SCHMIDT, KARL PATTERSON, B.A. (Cornell), Chief Curator of Zoology, *Chicago Natural History Museum, Chicago, Ill.*

SCHMIEDER, RUDOLF GUSTAV, A.B., A.M., Ph.D. (Pennsylvania), Assistant Professor of Zoology, *Zoological Laboratory, University of Pennsylvania, Philadelphia 4, Pa.*

SCHMITT, FRANCIS OTTO, A.B., Ph.D. (Washington University), Professor of Biology, Head of the Department of Biology and Biological Engineering, *Massachusetts Institute of Technology, Cambridge, Mass.*

SCHMITT, WALDO LA SALLE, B.A., Ph.D. (George Washington), M.A. (California), Head Curator Department of Biology, *U. S. National Museum, Washington, D. C.*

SCHNEIDER, BYRON AUBREY, A.B. (Western Maryland College), Sc.D. (Johns Hopkins), National Research Council Representative, Office of the Air Surgeon, *Hq. Army Air Forces, Washington, D. C.*

SCHNEIRLA, THEODORE CHRISTIAN, A.B., M.Sc., Sc.D. (Michigan), Associate Professor of Psychology, New York University, Associate Curator of Animal Behavior, *Department of Animal Behavior, American Museum of Natural History, Central Park West at 79th St., New York 24, N. Y.*

Scholander, Per Fredrik, Ph.D. (Oslo), Research Associate in Biology, *Martin Biological Laboratories, Swarthmore, Pa.*

Schooley, James Plummer, A.B., Ph.D. (Indiana), Assistant Professor of Anatomy, *Department of Anatomy, Wayne University College of Medicine, Detroit 26, Mich.*

Schotté, Oscar Emil, Licencié ès Sciences physiques et naturelles, D.Sc. (Geneva), M.A. (Amherst), Professor of Biology, *Amherst College, Amherst, Mass.*

Schrader, Franz, B.S., Ph.D. (Columbia), Professor of Zoology, *Columbia University, New York, N. Y.*

Schweizer, Malvina, B.S., M.S., Ph.D. (New York), Assistant Professor of Biology, Washington Square College, New York University, *50 West 67th St., New York, N. Y.*

Schwitalla, Alphonse Mary, A.B., A.M. (St. Louis), Ph.D. (Johns Hopkins), LL.D. (Tulane), D.Sc. (Lawrence College), Professor of Biology and Dean, St. Louis University School of Medicine, *1402 S. Grand Blvd., St. Louis, Mo.*

Scott, George G., A.B., A.M. (Williams), Ph.D. (Columbia), Professor of Biology, Emeritus, College of the City of New York, *460 Henkel Circle, Winter Park, Fla.*

Scott, Gordon Hatler, A.B. (Southwestern College, Kansas), M.A., Ph.D. (Minnesota), Professor of Anatomy and Chairman of the Department, *University of Southern California School of Medicine, Los Angeles 7, Calif.*

Scott, John Paul, A.B. (Wyoming), B.A. (Oxford), Ph.D. (Chicago), Research Associate and Chairman Division of Behavior Studies, *Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.*

Scott, John W., A.B., A.M. (Missouri), Ph.D. (Chicago), Professor of Zoology, Research Parasitologist, (Limited service), University of Wyoming, *1409 Garfield St., Laramie, Wyo.*

Seevers, Charles Hamilton, A.B. (Washburn College), Ph.D. (Chicago), Associate Professor of Zoology, *Roosevelt College of Chicago, 231 South Wells St., Chicago, Ill.*

Self, John Teague, B.A., M.A. (Baylor), Ph.D. (Oklahoma), Associate Professor of Zoology, *Department of Zoology, University of Oklahoma, Norman, Okla.*

Shapiro, Herbert, B.S., M.A. (Columbia), Ph.D. (Princeton), Instructor in Physiology, *Department of Physiology, Hahnemann Medical College, Philadelphia, Pa.*

Shaver, Jesse Milton, B.S.A. (Tennessee), M.S. (Vanderbilt), Ph.D. (Chicago), Professor of Biology, *George Peabody College for Teachers, Nashville, Tenn.*

Shelford, Victor Ernest, B.S., Ph.D. (Chicago), Professor of Zoology, University of Illinois, *Vivarium Building, Wright and Healey Sts., Champaign, Ill.*

Shettles, Landrum Brewer, A.B. (Mississippi College), M.S. (New Mexico), Ph.D., M.D. (Johns Hopkins), Instructor in Obstetrics, *The Johns Hopkins University and Hospital.* (On leave for military service, '44. 1st Lt. Medical Corps, U. S. A. 2827 Guilford Avenue, Baltimore 18, Md.)

Shoemaker, Hurst H., A.B. (Earlham), Ph.D. (Chicago), Associate in Zoology, University of Illinois, *Vivarium Building, Wright and Healey Sts., Champaign, Ill.*

Shull, A(aron) Franklin, A.B. (Michigan), Ph.D. (Columbia), Professor of Zoology, University of Michigan, *431 Highland Road, Ann Arbor, Mich.*

SHUMWAY, WALDO, A.B. (Amherst), A.M., Ph.D. (Columbia), Professor of Zoology, *317 N. H. Bldg., University of Illinois, Urbana, Ill.*

SICHEL, FERDINAND J. M., B.Sc. (McGill), Sc.M., Ph.D. (New York), Professor of Physiology, and Chairman of the Department, *College of Medicine, University of Vermont, Burlington, Vt.*

SIZER, IRWIN W(HITING), A.B. (Brown), Ph.D. (Rutgers), Associate Professor of Physiology, *Massachusetts Institute of Technology, Cambridge, Mass.*

SKOGSBERG, KARL JONAS TAGE, M.A., Ph.D. (Upsala, Sweden), Professor of Marine Biology and Oceanography, Stanford University, California, *Hopkins Marine Station, Pacific Grove, Calif.*

SLIFER, ELEANOR HEIST, B.S. (Temple), M.S., Ph.D. (Pennsylvania), Assistant Professor of Zoology, *Department of Zoology, University of Iowa, Iowa City, Iowa.*

SMALLWOOD, WILLIAM MARTIN, Ph.D., D.Sc. (Harvard), Professor of Comparative Anatomy, Emeritus, Syracuse University, *525 Euclid Ave., Syracuse, N. Y.*

SMELSER, GEORGE KEISER, A.B. (Earlham), Ph.D. (Chicago), Assistant Professor of Anatomy, *College of Physicians and Surgeons, Columbia University, 630 W. 168th St., New York, N. Y.*

SMITH, CHRISTIANNA, A.B. (Mount Holyoke), A.M., Ph.D. (Cornell), Professor of Zoology, *Mount Holyoke College, South Hadley, Mass.*

SMITH, DIETRICH CONRAD, A.B., A.M. (Minnesota), Ph.D. (Harvard), Associate Professor of Physiology, *University of Maryland, School of Medicine, Baltimore, Md.*

SNELL, GEORGE D., B.S. (Dartmouth), M.S., Sc.D. (Harvard), Research Associate, *Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.*

SNYDER, LAURENCE HASBROUCK, B.S. (Rutgers), M.S., Sc.D. (Harvard), Professor of Zoology and Chairman of the Department, *Department of Zoology, Ohio State University, Columbus, Ohio.*

SONNEBORN, TRACY MORTON, A.B., Ph.D. (Johns Hopkins), Professor of Zoology, *Indiana University, Bloomington, Ind.*

SPEICHER, BENJAMIN ROBERT, A.B. (Denison), M.S., Ph.D. (Pittsburgh), Associate Professor of Zoology and Acting Head, *Department of Zoology, University of Maine, Orono, Me.*

SPEIDEL, CARL CASKEY, Ph.B. (Lafayette), Ph.D. (Princeton), Sc.D. (Lafayette), Professor of Anatomy, *University of Virginia Medical School, University, Va.*

SPENCER, WARREN POPPINO, B.A. (Wooster), M.S., Ph.D. (Ohio State), Professor of Biology, *Wooster College, Wooster, Ohio.*

SPERRY, ROGER WOLCOTT, A.B., A.M. (Oberlin), Ph.D. (Chicago), Research Fellow in Biology, Harvard University and Yerkes Laboratory of Primate Biology, Consultant, Government Research Project OEMcmr-221, *Yerkes Laboratory of Primate Biology, Orange Park, Fla.*

SPRATT, NELSON TRACY, JR., A.B. (Emory), Ph.D. (Rochester), Research Assistant in Embryology and Instructor in Biology, *The Johns Hopkins University, Baltimore, Md.* (On leave for military service, '44. Assistant Director, *Altitude Training unit, Barksdale Field, La.*)

STABLER, ROBERT MILLER, A.B. (Swarthmore), Sc.D. (Johns Hopkins), Assistant Professor of Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia, Pa.*

Stanley, Willard Francis, A.B. (North Central), A.M., Ph.D. (Illinois), Head of the Science Department, *State Teachers College, Fredonia, N. Y.*

Steggerda, Frederic Russell, A.B. (Hope), M.A., Ph.D. (Minnesota), Associate Professor of Physiology, *416 Natural History Building, University of Illinois, Urbana, Ill.*

Steggerda, Morris, A.B. (Hope), A.M., Ph.D. (Illinois), Professor of Anthropology, *The Kennedy School of Missions, The Hartford Seminary Foundation, Hartford, Conn.*

Steinbach, Henry Burr, A.B. (Michigan), M.A. (Brown), Ph.D. (Pennsylvania), Associate Professor of Zoology, *Washington University, St. Louis, Mo.*

Steinberg, Arthur G., B.Sc. (College of the City of New York), M.A., Ph.D. (Columbia), Associate Professor of Biology, Antioch College and Chairman Department of Genetics, Fels Research Institute, *Fels Research Institute, Yellow Springs, Ohio.*

Steiner, Gotthold, Ph.D. (Berne), *Bureau of Plant Industry, U. S. Department of Agriculture, Washington, D. C.*

Stern, Curt, Ph.D. (Berlin), Professor of Experimental Zoology, Chairman, Division of Biological Sciences, *University of Rochester, Rochester, N. Y.*

Stier, Theodore James Blanchard, B.S. (Rutgers), M.A., Ph.D. (Harvard), Associate Professor of Physiology, *Indiana University School of Medicine, Bloomington, Ind.*

Stiles, Karl A., A.B. (Battle Creek), M.S., Ph.D. (Michigan), Professor of Biological Science, *The Basic College, Michigan State College, East Lansing, Mich.*

Stilwell, E. Frances, A.B., A.M. (Smith), Ph.D. (Bryn Mawr), Associate Professor of Anatomy, *Department of Anatomy, Woman's Medical College of Pennsylvania, Philadelphia, Pa.*

Stoll, Norman Rudolph, B.S. (Mount Union), M.S. (Michigan), Sc.D. (Johns Hopkins), Associate Member *Rockefeller Institute for Medical Research, Department of Animal Pathology, Princeton, N. J.*

Stone, R. G., Ph.D. (Missouri), Professor of Biology, *University of Kansas City, Kansas City, Mo.*

Stone, Wilson Stuart, A.B., M.A., Ph.D. (Texas), Professor of Zoology, *Department of Zoology and Physiology, University of Texas, Box 1797, University Station, Austin 12, Texas.*

Strandskov, Herluf Haldan, A.B. (Iowa State Teachers), M.S. (Illinois), Ph.D. (Chicago), Assistant Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Streeter, George Linius, A.B., Sc.D. (Union), A.M., M.D. (Columbia), Sc.D. (Dublin), LL.D. (Michigan), Research Associate, Carnegie Institution of Washington, *The Johns Hopkins Medical School, 710 North Washington St., Baltimore 5, Md.*

Strohecker, Henry Fred, A.B. (Mercer), Ph.D. (Chicago), Professor of Zoology, *Department of Zoology, New Mexico Highlands University, Las Vegas, N. M.*

Strong, Oliver S., A.B., A.M. (Princeton), Ph.D. (Columbia), Professor of Neurology and Neuro-Histology, Emeritus, College of Physicians and Surgeons, *630 W. 168th St., New York, N. Y.*

STRONG, REUBEN MYRON, A.B. (Oberlin), M.A., Ph.D. (Harvard), Professor of Anatomy, Loyola University School of Medicine, *706 South Wolcott St., Chicago 12, Ill.*

STUNKARD, HORACE W., B.S. (Coe), A.M., Ph.D. (Illinois), Professor of Biology, *New York University, University Heights, New York, N. Y.*

STURTEVANT, ALFRED HENRY, A.B., Ph.D. (Columbia), Professor of Genetics, *California Institute of Technology, Pasadena 4, Calif.*

SUMMERS, F(RANCIS) M(ARION), A.B. (California), M.A., Ph.D. (Columbia), Department of Biology, *College of the City of New York, New York, N. Y.*

SWEETMAN, HARVEY LEROY, B.S. (Colorado Agricultural), M.S. (Iowa State), Ph.D. (Massachusetts State), Assistant Professor of Entomology, *Massachusetts State College, Amherst, Mass.*

SWINGLE, WILBUR WILLIS, A.B., A.M. (Kansas), Ph.D. (Princeton), Professor of Biology, *Princeton University, Princeton, N. J.*

TALIAFERRO, WILLIAM HAY, B.S. (Virginia), Ph.D. (Johns Hopkins), Eliakim H. Moore Distinguished Service Professor of Parasitology, Chairman of the Department of Bacteriology and Parasitology; Adviser to the President in the Division of Biological Sciences, *The University of Chicago, 5724 Ellis Ave., Chicago 37, Ill.*

TAYLOR, AUBREY BRYANT, Ph.B. (Brown), M.S., Ph.D. (Iowa), Assistant Professor of Physiology, University of Illinois, *402 Natural History Building, Urbana, Ill.*

TAYLOR, CECIL A(LBERT), B.A. (Taylor), M.S. (Kentucky), Ph.D. (Chicago), Assistant Professor of Anatomy, *College of Dentistry, New York University, 477 First Ave., New York, N. Y.*

TAYLOR, CHARLES VINCENT, A.B. (Mount Morris), A.M., Ph.D. (California), Hertzstein Professor of Biology, *Stanford University, Calif.*

TAYLOR, GEORGE WELLFORD, B.A. (Richmond), M.A., Ph.D. (Princeton), Assistant Professor of Zoology, *Washington University, St. Louis, Mo.*

THOMAS, LYELL JAY, B.S. (Fargo), M.S., Ph.D. (Illinois), Associate Professor of Zoology, University of Illinois, *103 Vivarium Bldg., Champaign, Ill.*

TITTLER, IRVING ALBERT, B.S. (Bethany College), M.A., Ph.D. (Columbia), Assistant Professor of Biology. *Department of Biology, Brooklyn College, Bedford Ave. and Ave. H, Brooklyn, N. Y.*

TORREY, THEODORE WILLETT, A.B. (Denver), M.A., Ph.D. (Harvard), Associate Professor of Zoology, *Department of Zoology, Indiana University, Bloomington, Ind.*

TRAGER, WILLIAM, B.S. (Rutgers), M.A., Ph.D. (Harvard), Associate, Rockefeller Institute, *Department of Animal and Plant Pathology, Rockefeller Institute, Princeton, N. J.*

TREADWELL, AARON, L., B.S., M.S., Sc.D. (Wesleyan), Ph.D. (Chicago), Professor of Zoology, Emeritus, *Vassar College, Poughkeepsie, N. Y.*

TRESSLER, WILLIS LATTANNER, B.A., M.A., Ph.D. (Wisconsin), Assistant Professor of Zoology, Planktologist, Chesapeake Biological Laboratory, *Department of Zoology, University of Maryland, College Park, Md.*

TURNER, CLARENCE DONNELL, A.B. (Westminster), A.M., Ph.D. (Missouri), Associate Professor of Zoology, 105 Locy Hall, *Department of Zoology, Northwestern University, Evaston, Ill.*

TURNER, CARENCE LESTER, A.B., A.M. (Ohio Wesleyan), Ph.D. (Wisconsin), Professor of Zoology, *Northwestern University, Evanston, Ill.*

TWITTY, VICTOR CHANDLER, B.S. (Butler), Ph.D. (Yale), Professor of Biology, *Department of Biology, Stanford University, Palo Alto, Calif.*

TYLER, ALBERT, A.B., A.M. (Columbia), Ph.D. (California Institute of Technology), Assistant Professor of Embryology, *Department of Biology, California Institute of Technology, Pasadena 4, Calif.*

UNGER, WILLIAM BYERS, B.A., M.S., Ph.D. (Yale), M.A. (Hon.) (Dartmouth), Professor of Zoology, *Dartmouth College, Hanover, N. H.*

VAN CLEAVE, HARLEY JONES, B.S. (Knox), M.S., Ph.D. (Illinois), Professor of Zoology, *318 Natural History Building, University of Illinois, Urbana, Ill.*

VAN DER SCHALIE, HENRY, A.B. (Calvin), M.S., Ph.D. (Michigan), Curator of Mollusks and Assistant Professor of Zoology, University of Michigan, *Museum of Zoology, Ann Arbor, Mich.*

VAN DEVENTER, WILLIAM CARL, A.B. (Central), A.M., Ph.D. (Illinois), Teacher of Biology, *Division of Science, Stephens College, Columbia, Mo.*

VILLEE, CLAUDE ALVIN, JR., B.S. (Franklin and Marshall), Ph.D. (California), Assistant Professor of Biological Chemistry, *Department of Biological Chemistry, Harvard University Medical School, Boston, Mass.*

VISSCHER, J. PAUL, A.B. (Hope), A.M., Ph.D. (Johns Hopkins), Professor of Biology, *Western Reserve University, Cleveland, Ohio.*

WAGGENER, ROY ALFRED, B.A. (LaGrange), M.A. (Brown), Ph.D. (Cornell), Professor of Zoology, Chairman of the Department of Zoology, *Carleton College, Northfield, Minn.*

WAGNER, GEORGE, B.A. (Kansas), M.A. (Michigan), Professor of Zoology, Emeritus, University of Wisconsin, Biology Building, Madison, Wis., *546 Washington Ave., Palo Alto, Calif.*

WAITE, FREDERICK CLAYTON, Litt.B. (Adelbert), A.M., H.H.D. (Western Reserve), A.M., Ph.D. (Harvard), Professor of Histology and Embryology, Emeritus, Western Reserve University, *144 Locust St., Dover, N. H.*

WALLS, GORDON LYNN, B.S. (Tufts), A.M. (Harvard), Sc.D. (Michigan), Research Associate, *Department of Psychology, University of Rochester*, and Research Associate, *Scientific Bureau, Bausch and Lomb Optical Company, Rochester, N. Y.*

WALTON, ARTHUR CALVIN, B.A., M.A. (Northwestern), Ph.D. (Illinois), Wallace C. Abbott Professor of Biology, Chairman of the Department, *Knox College, Galesburg, Ill.*

WALZL, EDWARD MCCOLGAN, M.A., Ph.D., M.D. (Johns Hopkins), Assistant Professor of Otolaryngology, *The Johns Hopkins University, School of Medicine, Baltimore, Md.*

WANG, HSI, S.B., S.M. (Shanghai), Ph.D. (Chicago), Instructor in Anatomy, *Department of Zoology, University of Wisconsin, Madison, Wis.*

WANTLAND, WAYNE, W., B.S., A.M., Ph.D. (Northwestern), Co-Director of Biological Science, *Stephens College, Columbia, Mo.*

WARREN, ARTHUR EMERSON, B.A. (Acadia), Ph.D. (Harvard), Professor of Zoology, *McMaster University, Hamilton, Ontario, Canada.*

WARREN, DON C., A.B., A.M. (Indiana), Ph.D. (Columbia), Poultry Geneticist, *Kansas State College, Manhattan, Kan.*

Warwick, Bruce Lester, D.V.M. (Iowa State College), M.S., Ph.D. (Wisconsin), Animal Geneticist, *Division of Range Animal Husbandry, Texas Experiment Station, College Station, Texas.*

Watanabe, Yisamu, Rigakushi, Graduate of the Faculty of Natural Science, Imperial University of Japan, Rigakuhakushi (Sendai), Independent Investigator (Private Laboratory), *10, Nakajima Park, Sapporo, Japan.*

Waterman, Allyn Jay, A.B. (Oberlin), A.M. (Western Reserve), Ph.D. (Harvard), Associate Professor of Biology, *Williams College, Williamstown, Mass.*

Watterson, Ray Leighton, B.A. (Coe College), Ph.D. (Rochester), Assistant Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Weed, Lewis Hill, A.B. (Yale), M.D. (Johns Hopkins), Sc.D. (Rochester, Pennsylvania, Washington, Lafayette), LL.D. (Duke, Tufts, Tulane), Director of Medical School and Professor of Anatomy, *The Johns Hopkins Medical School, Baltimore 5, Md.*

Weese, Asa Orrin, A.B. (Minnesota), A.M., Ph.D. (Illinois), Professor of Zoology, *University of Oklahoma, Norman, Okla.*

Weichert, Charles K., B.S. (Rutgers), M.S., Ph.D. (Wisconsin), Professor of Zoology, *University of Cincinnati, Cincinnati, Ohio.*

Weill, Robert F., Sc.D. (Paris), Chef de Travaux à la Faculté des Sciences de Paris, *105 Boulevard Raspail, Paris VI, France.*

Weimer, Bernal R(obinson), A.B., A.M. (West Virginia), Ph.D. (Chicago), Professor of Biology and Dean of the Faculty, *Bethany College, Bethany, W. Va.*

Weinstein, Alexander, B.S., M.A., Ph.D. (Columbia), *Zoological Laboratory, Columbia University, New York, N. Y.*

Weiss, Paul Alfred, Ph.D. (Vienna), Professor of Zoology, Hull Zoological Laboratory, *The University of Chicago, Chicago 37, Ill.*

Weissenberg, Julius Richard, M.D. (Berlin), Professor of Histology and Embryology, *Middlesex University, School of Medicine, 54 Orange Street, Waltham 54, Mass.*

Welch, d'Alte Aldridge, M.A., Ph.D. (Johns Hopkins), Instructor of Biology, *John Carroll University, Cleveland 18, Ohio.*

Welch, Paul Smith, A.B. (James Millikin), A.M., Ph.D. (Illinois), Professor of Zoology, *University of Michigan, Ann Arbor, Mich.*

Wells, Lemen Jonathan, Ed.B. (Southern Illinois State Teachers College), A.M. (Northwestern), Ph.D. (Chicago), Associate Professor of Anatomy, *Department of Anatomy, University of Minnesota, Minneapolis, Minn.*

Welsh, John H., A.B. (Berea), M.A., Ph.D. (Harvard), Associate Professor of Zoology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

Wenner, William Franklin, B.A. (Lebanon Valley), Ph.D. (Yale), M.D. (Washington University, St. Louis), Medical Division, *The Upjohn Co., Kalamazoo, Mich.*

Wenrich, David Henry, B.A., M.A. (Kansas), Ph.D. (Harvard), Professor of Zoology, *University of Pennsylvania, Philadelphia 4, Pa.*

Wharton, George Willard, Jr., B.S., Ph.D. (Duke), Instructor in Zoology, *Zoology Department, Duke University, Durham, N. C.* (On leave for military service, '44).

WHEDON, ARTHUR D., B.A. (Iowa), Ph.D. (Pennsylvania), Professor of Zoology, *North Dakota Agricultural College, Fargo, N. D.*

WHITAKER, DOUGLAS M(ERRITT), *A.B.*, Ph.D. (Stanford), Professor of Biology, *Stanford University, Stanford University, Calif.*

WHITING, ANNA RACHEL (Mrs.), A.B. (Smith), Ph.D. (Iowa), Associate Professor of Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia 4, Pa.*

WHITING, PHINEAS WESCOTT, A.B., M.S. (Harvard), Ph.D. (Pennsylvania), Associate Professor of Zoology, *University of Pennsylvania, Philadelphia 4, Pa.*

WHITNEY, DAVID DAY, B.A. (Wesleyan), M.A., Ph.D. (Columbia), Chairman, Department of Zoology, *University of Nebraska, Lincoln, Nebr.*

WICHTERMAN, RALPH, B.S. (Temple), M.A., Ph.D. (Pennsylvania), Assistant Professor of Biology, *Biology Department, Temple University, Philadelphia, Pa.*

WIEMAN, HARRY LEWIS, A.B., A.M. (Cincinnati), Ph.D. (Chicago), Professor of Zoology, *University of Cincinnati, Cincinnati, Ohio.*

WILBER, CHARLES GRADY, B.Sc. (Marquette), M.A., Ph.D. (Johns Hopkins), Assistant Professor of General Physiology, *Department of Physiology, Fordham University, New York, N. Y.*

WILLARD, W. A., A.B. (Grinnell), A.M. (Tufts, Harvard), Ph.D. (Harvard), Professor of Anatomy, *University Medical College of Nebraska, 42nd St. and Dewey Ave., Omaha, Nebr.*

WILLEY, CHARLES HERBERT, A.B., ScM., Ph.D. (New York), Associate Professor of Biology, *New York University, University Heights, N. Y.*

WILLIAMS, CARROLL MILTON, B.S. (Richmond), A.M., Ph.D. (Harvard), Junior Prize Fellow in Biology, *Biological Laboratories, Harvard University, Cambridge, Mass.*

WILLIAM, STEPHEN RIGGS, A.B., A.M. (Oberlin), A.M., Ph.D. (Harvard), Professor of Zoology, Emeritus, Miami University, *300 East Church St., Oxford, Ohio.*

WILLIER, BENJAMIN H(ARRISON), B.S., Sc.D. (Wooster), Ph.D. (Chicago), Henry Walters Professor of Zoology, Chairman of the Department of Biology, *Department of Biology, The Johns Hopkins University, Baltimore 18, Md.*

WILSON, FRANCIS HENRY, B.S., M.S., Ph.D. (Cornell), Associate Professor of Parasitology, Department of Tropical Medicine, and Associate Professor of Zoology, *Department of Zoology, Tulane University, New Orleans, La.*

WILSON, IRA TEMPLIN, A.B., A.M., Ph.D. (Indiana), Professor of Biology, *Heidelberg College, Tiffin, Ohio.*

WILSON, J(AMES) WALTER, Ph.B., Ph.D. (Brown), Frank L. Day Professor of Biology, Brown University, *282 Doyle Ave., Providence, R. I.*

WISLOCKI, GEORGE BERNAYS, A.B. (Washington), M.D. (Johns Hopkins), James Stillman Professor of Comparative Anatomy, *Harvard Medical School, 25 Shattuck St., Boston, Mass.*

WITSCHI, EMIL, Ph.D. (Munich), Professor of Zoology, (Experimental Embryology), *State University of Iowa, Iowa City, Iowa.*

WODSEDALEK, JERRY EDWARD, Ph.B., M.Ph., Ph.D. (Wisconsin), Professor of Zoology, *University of Minnesota, Minneapolis, Minn.*

WOLF, ERNST, Ph.D. (Heidelberg), Research Associate, *Biological Laboratories, Harvard University, Divinity Ave., Cambridge, Mass.*

Wolfe, Harold R(eclus), B.S. (Miami, Florida), M.S. (Rutgers), Ph.D. (Wisconsin), Associate Professor of Zoology, *Biology Building, University of Wisconsin, Madison, Wis.*

Woodside, Gilbert Llewellyn, A.B. (DePauw), A.M., Ph.D. (Harvard), Assistant Professor of Biology, *Department of Entomology and Zoology, Massachusetts State College, Amherst, Mass.*

Woodruff, Lorande Loss, A.B., Ph.D. (Columbia), M.A. (Hon.) (Yale), Colgate Professor of Protozoology and Director of the Osborn Zoological Laboratory, *Yale University, New Haven, Conn.*

Woodward, Alvalyn E., Ph.B., M.S. (Rochester), Ph.D. (Michigan), Assistant Professor of Zoology, *University of Michigan, Ann Arbor, Mich.*

Worley, Leonard George, A.B., M.A. (Nebraska), Ph.D. (Harvard), Associate Professor, *Department of Biology, Brooklyn College, N. Y.*

Wright, Albert Hazen, A.B., A.M., Ph.D. (Cornell), Professor of Zoology, Cornell University, *113 E. Upland Road, Ithaca, N. Y.*

Wright, Sewall, B.S. (Lombard), M.S. (Illinois), Sc.D. (Harvard), Ernest D. Burton Distinguished Service Professor of Zoology, *Hull Zoological Laboratory, The University of Chicago, Chicago 37, Ill.*

Wyman, Jeffries, Jr., A.B. (Harvard), Ph.D. (University College, London), Associate Professor of Zoology, *Biological Laboratories, Harvard University, Divinity Ave., Cambridge, Mass.*

Yeager, J(ames) Franklin, Ph.B. (Yale), M.A. (Columbia), Ph.D. (New York), Senior Entomologist, *Bureau of Entomology and Plant Quarantine, Bldg. A., Beltsville, Md.*

Yocom, Harry, Barclay, A.B. (Oberlin), M.A., Ph.D. (California), Professor of Zoology, *University of Oregon, Eugene, Ore.*

Young, Benjamin P., B.S. (Kansas), Ph.D. (Cornell), Associate Professor of Zoology, Cornell University, *117 Parkway, Ithaca, N. Y.*

Young, R. T., B.S. (Pennsylvania), Ph.D. (Nebraska), *Scripps Institution of Oceanography, La Jolla, Calif.*

Young, William Caldwell, A.B., A.M. (Amherst), Ph.D. (Chicago), Associate Professor of Anatomy, *Department of Anatomy, University of Kansas, Lawrence, Kan.*

Zangerl, Rainer, Ph.D. (Zurich), Assistant Professor of Biology, *University of Notre Dame, Notre Dame, Ind.*

Zirkle, Raymond E., A.B., Ph.D. (Missouri), Professor of Botany, Director, *Institute of Radiobiology and Biophysics, The University of Chicago, Chicago 37, Ill.*

ASSOCIATE MEMBERS

Alexander, Lloyd Ephraim, A.M. (Michigan), Ph.D. (Rochester), Assistant Professor of Biology, *Fisk University, Nashville, Tenn.*

Altland, Paul Daniel, B.S. (Gettysburg), M.A., Ph.D. (Duke), Assistant Professor of Biology, *Department of Biology, Gettysburg College, Gettysburg, Pa.* (On leave for military service, '44. Physiologist, *U.S. Public Health Service, National Institute of Health, Bethesda, Md.*)

ASHKENAZ, ELEANOR WEINER, B.S., Ph.D. (Pennsylvania), Research Associate in Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia 4, Pa.*

BAIER, JOSEPH GEORGE, JR., B.S., M.S. (Rutgers), Ph.D. (Wisconsin), Professor of Zoology, Milwaukee Extension Center, University of Wisconsin, *623 W. State St., Milwaukee, Wis.*

BAILEY, JOHN WENDELL, B.S., M.S. (Mississippi State), A.B., A.M. (Cornell), A.M., Ph.D. (Harvard), Professor of Biology, University of Richmond, *27 Willway Road, Richmond 21, Va.*

BAILEY, SARAH WATSON, B.S., M.S. (Middlebury), A.M., Ph.D. (Radcliffe), *15 Campden Road, Scarsdale, N. Y.*

BAKER, CLINTON L(YLE), B.S., M.S. (Emory), Ph.D. (Columbia), Professor of Biology, *Department of Biology, Southwestern College, Memphis, Tenn.*

BAKER, E(DGAR) G(ATES) STANLEY, A.B. (De Pauw), Ph.D. (Stanford), Research Assistant in Biology, *Department of Biology, Stanford University, Stanford University, Calif.* (On leave for military service, 1st Lt. *Air Force, A.U.S., Altitude Training Unit, C.A.A.B., Columbia, S. C.*)

BALLARD, WILLIAM WHITNEY, B.S. (Dartmouth), Ph.D. (Yale), Professor of Zoology and Embryology, *Dartmouth College, Hanover, N. H.*

BARRETT, W(ILLIAM) C(LARK), JR., B.S., M.S. (Virginia), Ph.D. (Harvard), Assistant Professor of Histology and Embryology, *School of Medicine, Western Reserve University, Cleveland, Ohio.*

BARRY, ALEXANDER, A.B., M.A., Ph.D. (Harvard), Instructor in Anatomy, *Department of Anatomy, University of Michigan, Ann Arbor, Mich.*

BENNETT, MARY ALLISON, B.S. (Western Illinois State Teachers College), A.M., Ph.D. (Chicago), Associate Professor of Biology, *Western Illinois State Teachers College, Macomb, Ill.*

BENNINGTON, NEVILLE L(YNNE), B.A. (Wooster), M.A., Ph.D. (Northwestern), Associate Professor of Biology, *Department of Biology, Beloit College, Beloit, Wis.*

BERNARD, RICHARD, B.Sc., M. Sc. (Laval University, Quebec), Ph.D. (Cornell), Associate Director, Quebec Zoological Garden, Charlesbourg, Quebec, Instructor in Physiology, Department of Biology, Laval University, *Quebec Zoological Garden, Charlesbourg, Quebec, Canada.*

BERRY, LEVETTE JOE, B.S. (Southwest Texas Teachers), Ph.D. (Texas), Assistant Professor of Biology, *Dalton Hall, Bryn Mawr College, Bryn Mawr, Pa.*

BERRY, RAYMOND ORVIL, B.S. (North Texas State Teachers College), M.S. (Agricultural and Mechanical College of Texas), Ph.D. (Johns Hopkins), Associate Geneticist, Division of Range Animal Husbandry, *Texas Agricultural Experiment Station, College Station, Texas.*

BISHOP, MABEL LOWELL, A.B. (Wellesley), A.M. (Smith), Ph.D. (Chicago), Professor of Zoology and Head of the Department, *Department of Biology, Hood College, Frederick, Md.*

BLACK, EDGAR CLARK, B.A. (McMaster), M.A. (British Columbia), Ph.D. (Pennsylvania), Assistant Professor of Physiology, *Department of Physiology, Dalhousie University, Halifax, Nova Scotia, Canada.*

BLAIR, ALBERT PATRICK, B.S. (Tulsa), Ph.D. (Indiana), *Laboratory of Experimental Biology, The American Museum of Natural History, New York, N. Y.*

BLAKE, IRVING H(ILL), A.B. (Bates), A.M. (Brown), Ph.D. (Illinois), Professor of Zoology and Anatomy, *Department of Zoology, University of Nebraska, Lincoln, Nebr.*

BLANC, RICHARD, A.B., M.A. (Amherst), Ph.D. (California), Instructor in Zoology, *Department of Zoology, University of Rochester, Rochester, N. Y.*

BLIVAISS, BEN B(URTON), B.S., M.S., Ph.D. (Chicago), Instructor in Physiology, *Department of Zoology and Physiology, Natural History Museum, University of Illinois, Urbana, Ill.*

BLOUNT, ISABEL HARPER, A.B., M.A. (Smith), Ph.D. (Yale), Voluntary Research Worker, *Department of Anatomy, University of Texas, Medical Branch, Galveston, Texas.*

BOCHE, ROBERT DEVORE, B.S., Ph.D. (California Institute of Technology), Instructor in Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia, Pa.*

BOLING, JOHN LANDRUM, A.B. (Linfield), Ph.D. (Brown), Instructor in Anatomy, *New York University College of Medicine, 477 First Ave., New York 16, N. Y.*

BONNET, DAVID DUDLEY, B.S., M.A., Ph.D. (Harvard), Assistant Sanitarian (R) USPHS, Acting Director, Dengue Mosquito Control, *Territorial Board of Health, Honolulu, Hawaii.*

BOTSFORD, E. FRANCES, B.A. (Mount Holyoke), M.S., Ph.D. (Yale), Associate Professor of Zoology, *Connecticut College, New London, Conn.*

BOWEN, R. EARL, A.B. (Hendrix College), A.M., Ph.D. (Harvard), Professor of Biology, Head of the Department of Biology, *Gettysburg College, Gettysburg, Pa.*

BOWEN, WILLIAM J., A.B., M.A. (Buffalo), Ph.D. (Johns Hopkins), *Department of Zoology, University of North Carolina, Chapel Hill, N. C.*

BRADDOCK, JAMES CONGER, A.B. (Williams), Ph.D. (Chicago), Assistant Professor of Zoology, *Department of Zoology, University of Idaho, Moscow, Idaho.*

BREHME, KATHERINE SUYDAM, A.B. (Barnard), A.M. (Virginia), Ph.D. (Columbia), Lecturer in Anatomy, *Department of Anatomy, Cornell University Medical College, 1300 York Avenue, New York, N. Y.*

BRIGGS, ROBERT WILLIAM, B.S. (Boston), Ph.D. (Harvard), Biologist, *Lankenau Hospital Research Institute, Philadelphia, Pa.*

BRYSON, VERNON, A.B. (California), M.A., Ph.D. (Columbia), Physiologist (Army contract), *Biological Laboratory, Long Island Biological Association, Cold Spring Harbor, Long Island, N. Y.*

BUNDE, CARL ALBERT, B.S., M.A., Ph.D. (Wisconsin), Assistant Professor of Physiology and Pharmacology, *Southwestern Medical College, 2311 Oaklawn Ave., Dallas 4, Texas.*

BURBANCK, WILLIAM DUDLEY, B.A. (Earlham), M.S. (Haverford), Ph.D. (Chicago), Associate Professor of Biology and Chairman of the Department, *Department of Biology, Drury College, Springfield, Mo.*

BURDICK, HAROLD ORMOND, B.A. (Milton College), M.A. (Wisconsin), Sc.D. (Salem College), Professor of Biology, Alfred University, *17 Sayles St., Alfred, N. Y.*

BUSCH, KARL H. D., B.S. (Capital), M.S., Ph.D. (Ohio), Professor of Biology and Chairman of the Departments of Biology and Nursing Education, *Department of Biology, Augustana College, Sioux Falls, S. D.*

BUSHNELL, RALPH JUDSON, B.S., M.S., Ph.D. (Wisconsin), Assistant Professor of Genetics, *University of Connecticut, Storrs, Conn.*

BUTLER, ELIZABETH, A.B. (Vassar), Ph.D. (Chicago), *90 Commonwealth Ave., Boston, Mass.*

BUTT, FERDINAND HINCKLEY, A.B., M.A. (Washington), Ph.D. (Cornell), Assistant Professor of Insect Morphology, Histology and Embryology, *Cornell University, Ithaca, N. Y.*

CABLE, LOUELLA E., A.B., A.M. (South Dakota), Aquatic Biologist, *U. S. Fish and Wildlife Service, College Park, Md.*

CALDWELL, GEORGE THORNHILL, B.S. (William and Mary), S.M., Ph.D. (Chicago), Professor of Zoology, *University of Arizona, Tucson, Ariz.*

CAMPBELL, ROBERT SEYMOUR, A.B., M.A. (Saskatchewan), Ph.D. (Michigan), Assistant Professor of Zoology, *109 Wildlife Conservation Building, University of Missouri, Columbia, Mo.*

CARLSON, LOREN DANIEL, B.S. (St. Ambrose College), Ph.D. (Iowa), Instructor in Physiology, *Department of Physiology, University of Washington, School of Medicine, Seattle 5, Wash.*

CARROLL, PAUL L., S.J., A.B., M.A., M.S., Ph.D. (St. Louis), Associate Professor of Biology, *St. Louis University, School of Medicine, 1402 S. Grand Blvd., St. Louis, Mo.*

CASTLE, GORDON BENJAMIN, A.B. (Wabash College), M.A., Ph.D. (California), Professor of Zoology and Chairman of the Department of Zoology, *Montana State University, Missoula, Mont.*

CHASE, ELIZABETH BROWN, A.B., A.M. (Minnesota), Ph.D. (Chicago), Guest Investigator, *Department of Zoology, University of Illinois, Urbana, Ill.*

CHASE, HYMAN YATES, B.Sc., M.Sc. (Howard), Ph.D. (Stanford), Associate Professor of Zoology, *Department of Zoology, Howard University, 654 Girard St., N. W., Washington, D. C.*

CHESTER, WEBSTER, A.B. (Colgate), A.M. (Harvard), Sc.D. (Colgate), Professor of Biology and Head of the Department, *Colby College, Waterville, Me.*

CHURNEY, LEON, A.B., Ph.D. (Pennsylvania), Instructor in Zoology, *University of Pennsylvania, Philadelphia, Pa.*

CIESLAK, EDWIN S., B.S. (Chicago), M.S. (Northwestern), Ph.D. (Chicago), Assistant Professor of Biology, *University of Minnesota, The General College, Minneapolis, Minn.*

CLANCY, CLARENCE WILLIAM, B.S., M.S. (Illinois), Ph.D. (Stanford), Assistant Professor of Biology, *Department of Biology, University of Oregon, Eugene, Ore.*

CLARK, HUGH, B.A. (Clark), Ph.D. (Michigan), Associate in Zoology, *Department of Zoology, State University of Iowa, Iowa City, Ia.*

CLARKE, L. FLOYD, B.S., M.S. (Utah State Agricultural College), Ph.D. (Chicago), Associate Professor of Zoology and Chairman of the Department of Zoology, Director, Pre-medical Curriculum, University of Wyoming, *Laramie, Wyo.*

Cole, La Mont C., S.B. (Chicago), M.S. (Utah), Ph.D. (Chicago), Instructor in Zoology, Department of Zoology, Indiana University, Bloomington, Ind.

Collias, Nicholas Elias, S.B., Ph.D. (Chicago), Instructor in Biology, *Department of Biology, Amherst College, Amherst, Mass.*

Colwin, Arthur Lentz, B.Sc., M.Sc., Ph.D. (McGill), Instructor in Biology, *Queens College, Flushing, N. Y.*

Cooper, Ruth Snyder, A.B., M.A., Ph.D. (Columbia), Research Assistant, *Department of Zoology, Princeton University, Princeton, N. J.*

Copeland, Donald Eugene, A.B. (Rochester), M.A. (Amherst), Ph.D. (Harvard), Instructor in Biology, *Department of Biology, Brown University, Providence, R. I.*

Crosman, Arthur Marston, B.S. (Dartmouth), A.M. (Columbia), Ph.D. (Cornell), Assistant Professor of Biology, *Washington Square College, New York University, New York, N. Y.*

Crouse, Helen V(irginia), A.B. (Goucher), A.M. (Smith), Ph.D. (Missouri), Research Assistant in Zoology, *Department of Zoology, University of Pennsylvania, Philadelphia, Pa.*

Crowell, Prince Sears, Jr., A.B. (Bowdoin), A.M., Ph.D. (Harvard), Assistant Professor of Zoology, *Miami University, Oxford, Ohio.*

Dakin, Janet Wilder, (Mrs. W. S.) B.A., M.A. (Mount Holyoke), Ph.D. (Chicago), *54 Snell St., Amherst, Mass.*

Dalton, Howard Clark, B.A., M.A. (Wesleyan), Ph.D. (Stanford), Instructor in Biology, *Department of Biology, Brown University, Providence 12, R. I.*

D'Angelo, Savino Albert, A.B., M.S., Ph.D. (Washington Square College), Instructor in Biology, *Washington Square College, New York University, 100 Washington Square East, New York, N. Y.*

Daniel, George Edwin, B.S. (Arkansas), Sc.D. (Johns Hopkins), Research Fellow, *National Cancer Institute, U. S. Public Health Service, Bethesda, Md.*

Danielson, Ragnar Nathanael, B.A., M.A., Ph.D. (Minnesota), *Department of Physiology, Medical College of the State of South Carolina, Charleston 16, S. C.*

Davenport, Demorest, A.B., Ph.D. (Harvard), M.A. (Colorado College), Assistant Professor of Biology, *Reed College, Portland, Ore.* (On leave for military service, '42. 2nd Lt., U. S. A.)

Day, Maxwell Frank Cooper, B.Sc. (Sydney, Australia), A.M., Ph.D. (Harvard), *Australian Legation, Scientific Research Liaison Office, Room 6, Dupont Circle Bldg., Washington 9, D. C.*

Degener, Lyda May, A.B., A.M., Ph.D. (Cornell), *"Twin Acres," Ivyland, Bucks Co., Pa.*

DeLanney, Louis Edgerton, B.A., M.A. (California), Ph.D. (Stanford), Instructor in Physiology, *San Jose State College, San Jose, Calif.*

Dennis, Earl Aubrey, B.A. (Wooster), Ph.D. (Chicago), Professor of Biology, *American University, Washington, D. C.*

Derbyshire, Russel Clay, B.Sc. (Kansas State), Ph.D. (Iowa State), Instructor in Zoology and Comparative Anatomy, *University of Omaha, Omaha, Nebr.*

DERICKSON, SAMUEL HOFFMAN, B.S., M.S., S.D. (Lebanon Valley), Professor of Biological Science and Head of the Department, *Tyrone Biological Laboratory, Lebanon Valley College, Annville, Pa.*

DILDINE, GLENN C(RAWFORD), A.B. (De Pauw), M.S., Ph.D. (Northwestern), Chairman, Division of Science and Mathematics, *State Teachers College, Moorhead, Minn.*

DOBROVOLNY, CHARLES GEORGE, A.B. (Montana), M.S. (Kansas State College), Ph.D. (Michigan), Assistant Professor, Department of Zoology, *University of New Hampshire, Durham, N. H.*

DORNFELD, ERNST JOHN, B.S. (Marquette), M.A., Ph.D. (Wisconsin), Associate Professor of Zoology, *Department of Zoology, Oregon State College, Corvallis, Ore.*

DOUGHERTY, ELLSWORTH CHARLES, A.B., Ph.D., M.D. (California), Research Fellow in Zoology and Physiologist, Donner Laboratory of Medical Physics, University of California, *Department of Zoology, University of California, Berkeley 4, Calif.*

DROPKIN, VICTOR H., B.A. (Cornell), Ph.D. (Chicago), Instructor in Zoology in the College, *The University of Chicago, Chicago 37, Ill.* (On leave for military service, '43. 2nd Lt. Sanitary Corps.)

DRURY, HORACE FEATHERSTONE, B.S. (George Washington), A.M., Ph.D. (Harvard), Tutor in Biology, *Brooklyn College, Brooklyn, N. Y.* (On leave for military service, '43. *Lt. A. C. Altitude Training Unit, Army Air Base, Ephrata, Wash.*)

DUGAL, LOUIS PAUL, B.A., M.A., M.Sc. (Laval), Ph.D. (Pennsylvania), Professeur agrégé de Physiologie générale, Université de Montréal, *2806 Maplewood, Montreal, Quebec, Canada.*

DUNHAM, H(ENRY) HOWARD, B.S. (Kansas State Teachers), Sc.M., Ph.D. (Brown), Assistant Instructor, *Department of Anatomy, University of Kansas, Lawrence, Kan.*

DUNKLE, DAVID HOSBROOK, A.B. (Kansas), Ph.D. (Harvard), Assistant in charge of paleontology, *Department of Paleontology, Cleveland Museum of Natural History, 2717 Euclid Ave., Cleveland, Ohio.*

DURYEE, WILLIAM RANKIN, B.A., Ph.D. (Yale), *National Research Council, 2101 Constitution Ave., Washington 25, D. C.*

EADIE, WILLIAM ROBERT, B.S., M.S. (New Hampshire), Ph.D. (Cornell), Assistant Professor of Zoology, *Department of Zoology, Cornell University, Ithaca, N. Y.*

EDDY, MILTON W., B.S., M.S. (Northwestern), Ph.D. (Pennsylvania), Professor of Biology, Dickinson College, *249 W. Louther St., Carlisle, Pa.*

EDMONDSON, W(ALLES) T(HOMAS), B.S., Ph.D. (Yale), *Woods Hole Oceanographic Institute, Woods Hole, Mass.*

EIGENBRODT, HAROLD JOHN, B.A. (North Central), M.A., Ph.D. (Illinois), Professor of Zoology, North Central College, *631 E. North Ave., Naperville, Ill.*

EVERETT, NEWTON BENNIE, B.S., M.S. (North Texas State Teachers College), Ph.D. (Michigan), Assistant Professor of Anatomy, *Department of Anatomy, University of Washington, School of Medicine, Seattle 5, Wash.*

Ferguson, Frederick Palmer, B.A., M.A. (Wesleyan), Ph.D. (Minnesota), Research Associate in Biology, *Bureau of Biological Research, Rutgers University, New Brunswick, N. J.*

Ferguson, Malcolm Stuart, B.A., M.A. (Western Ontario), Ph.D. (Illinois), Assistant, Rockefeller Institute. (On leave for military service, '42, Capt. U. S. A., Sanitary Corps, *APO, 468 c/o Postmaster, San Francisco, Calif.*

Flemister, Sarah Elizabeth, A.B., A.M., Ph.D. (Duke), *Box 463, Balboa Island, Calif.*

Flynn, Carl Munro, A.B. (Maine), M.A. (Wesleyan), M.A., Ph.D. (Harvard), Instructor in Zoology, *University of Maine, Orono, Me.*

Forbes, Grace Springer, A.B., A.M. (Oberlin), Ph.D. (Columbia), Instructor in Zoology, *Bard College, Annandale-on-Hudson, N. Y.*

Foster, Kendall Wilson, B.S. (Tufts), A.M., Ph.D. (Harvard), Instructor in Science, *Groton School, Groton, Mass.*

Foster, Mark A., A.B. (Louisiana Normal), M.S. (Kansas State Agricultural College), Ph.D. (Wisconsin), M.D. (Harvard), Fellow in Medicine, *Mayo Clinic, Rochester, Minn.* (On leave for military service, '42. Lt. (j.g.), U. S. N. R. *6662 Hillandale Road, Bethesda, Md.*)

Foulks, James Grigsby, B.A. (Rice), Ph.D. (Johns Hopkins), Instructor in Anatomy, *Department of Anatomy, Ohio State University, Columbus, Ohio.*

Frizzell, Harriet Exline, A.B. (Reed), M.S., Ph.D. (Washington), *Apartodo 803, c/o International Petroleum Company, Guayaquil, Ecuador, South America.*

Fry, Frederick Ernest Joseph, M.A., Ph.D. (Toronto), Lecturer and Research Assistant, *Department of Biology, University of Toronto, Toronto, Canada.*

Furrow, Clarence Lee, A.B., A.M. (Oklahoma), Ph.D. (Iowa), Professor of Biology, *Knox College, Galesburg, Ill.*

Gabriel, Mordecai L., B.A. (Yeshiva), M.A., Ph.D. (Columbia), Research Assistant in Genetics, *Storrs Agricultural Experiment Station, Storrs, Conn.*

Gatz, Arthur John, B.A. (Carleton), M.A., Ph.D. (Minnesota), Assistant Professor of Anatomy, *Loyola University School of Medicine, 706 So. Wolcott, Chicago 12, Ill.*

Gayer, H. Kenneth, A.B. (Oberlin), Ph.D. (Washington), Special Lecturer in Physics and Instructor in Zoology, *Department of Biology, Grinnell College, Grinnell, Iowa.*

George, Ira D., B.S., M.S. (Louisiana State), Ph.D. (Michigan), Associate Professor of Zoology, *Department of Zoology, Box 8729, Louisiana State University, Baton Rouge 3, La.*

Gier, Herschel T., A.B. (Kansas State Teachers), Ph.D. (Indiana), Assistant Professor of Zoology, *Ohio University, Athens, Ohio.*

Gilbert, Perry Webster, A.B. (Dartmouth), Ph.D. (Cornell), Assistant Professor of Zoology, *Department of Zoology, Cornell University, Stimson Hall, Ithaca, N. Y.*

Goodchild, Chauncey G., B.S. (Westminster College), Ph.D. (New York), Professor of Biology, *Department of Biology, Southwest Missouri State Teachers College, Springfield, Mo.*

Granger, Barbara Samson, A.B., A.M. (Mount Holyoke), Ph.D. (Cornell), Instructor in Zoology, *Department of Zoology, University of Arizona, Tucson, Ariz*

Grant, Ronald, B.Sc. (London), Ph.D. (Edinburgh), Lecturer in Zoology, McGill University, *Department of Zoology, McGill University, Montreal, Quebec, Canada.*

Gray, Stephen Wood, A.B. (Lake Forest), M.A., Ph.D. (Illinois), Instructor in Physiology, *427 Natural History Building, University of Illinois, Urbana, Ill.* (On leave for military service, '44. *Section E-2 Tyndall Field, Fla.*)

Green, Earl Leroy, B.S. (Allegheny College), ScM., Ph.D. (Brown), Instructor in Zoology, *Department of Zoology and Entomology, Ohio State University, Columbus, Ohio.* (On leave for military service, '43, 2nd Lt. U. S. A. Air Force.)

Green, Elizabeth Ufford (Mrs. Louis Craig Green), A.B., M.A., Ph.D. (Bryn Mawr), Biologist, *Lankenau Hospital Research Institute, Girard and Corinthian Aves, Philadelphia, Pa.*

Greiff, Donald, B.S. (Marquette), Sc.D. (Johns Hopkins), Assistant Professor of Biology, *Department of Biology, Saint Louis University, 1402 South Grand Blvd., St. Louis, Mo.*

Grobstein, Clifford, B.S., (College of the City of New York), M.S., Ph.D. (California), Instructor in Zoology, *Department of Zoology, Oregon State College, Corvallis, Ore.*

Hadidian, Zareh, B.S., M.S. (Rensselaer), Ph.D. (Clark), Instructor in Biology, *Wooster Foundation for Experimental Biology, 222 Maple Ave., Shrewsbury, Mass.*

Hagquist, Carl Waldemar, Ph.B., Ph.D. (Brown), Associate Professor of Biology, *Department of Biology, University of Richmond, Richmond, Va.*

Hall, B(enjamin) Vincent, A.B. (Linfield), Ph.D. (Yale), Assistant Professor of Zoology, University of Illinois, *Natural History Building, Urbana, Ill.*

Hall, Sam Rutherford, A.B. (Washington and Lee), A.M., Ph.D. (Virginia), *Bureau of Dairy Industry, U. S. Department of Agriculture, Beltsville, Md.*

Hammond, Datus Miller, B.S. (Utah State Agricultural College), M.A., Ph.D. (California), Associate Protozoologist, *U. S. Regional Laboratory for Animal Disease Research, Auburn, Ala.*

Hansborough, Louis Armstead, S.B. (Harvard), M.S. (Chicago), Ph.D. (Harvard), Assistant Professor of Zoology, *Howard University, Washington, D. C.*

Hansen, Ira Bowers, B.S., M.A. (Wesleyan), Ph.D. (Chicago), Professor of Zoology, *Department of Zoology, George Washington University, Washington, D. C.*

Hardcastle, Aaron Bascom, B.A., M.A. (Richmond), Ph.D. (Duke), Ensign, H-V (S) U. S. N. R., Specialist, Medical Corps, U. S. Navy, *4330 Clagett Road, University Park, Hyattsville, Md.*

Hardin, Garrett (James), Sc.B. (Chicago), Ph.D. (Stanford), Lecturer, *Science Department, Santa Barbara College, Santa Barbara, Calif.*

Harman, Pinckney Jones, B.S., M.S. (Georgetown), Ph.D. (Yale), Instructor in Anatomy, *Department of Anatomy, Georgetown University, School of Medicine, Washington, D. C.*

HAUBER, ULRICH ALBERT, A.B. (St. Ambrose), M.A. (Catholic), Ph.D. (Iowa), Professor of Biology, *St. Ambrose College, Davenport, Iowa.*

HEFLEY, HAROLD MARTIN, B.S., M.S., Ph.D. (Oklahoma), Medical Technician, *400 N.W. 10th St., Oklahoma City, Okla.*

HENDEE, ESTHER C., A.B. (Oberlin), Ph.D. (California), Associate Professor of Biology, *Russell Sage College, Troy, N. Y.*

HEWITT, WILLIAM FRANCIS, JR., A.B. (Princeton), S.M., Ph.D. (Chicago), Assistant Professor of Zoology, *Department of Zoology, University of Wichita, Wichita, Kan.*

HIGBEE, EDNA, B.S., M.S., Ph.D. (Pittsburgh), Head of Biology, Vice-Principal University School, *2845 Broadway Avenue, South Hills Station, Pittsburgh, Pa.*

HITCHCOCK, HAROLD BRADFORD, B.A. (Williams College), M.A., Ph.D. (Harvard), Assistant Professor of Biology, *Middlebury College, Middlebury, Vt.*

HOLT, VESTA, A.B. (Oregon), M.A. (Columbia), Ph.D. (Stanford), Professor of Biological Sciences and Head of the Department, *Ohio State College, Chico, Calif.*

HORN, EDWARD CHARLES, B.S. (Trinity College), A.M., Ph.D. (Princeton), Instructor in Zoology, *Department of Zoology, Duke University, Durham, N. C.*

HOROWITZ, NORMAN H., B.S. (Pittsburgh), Ph.D. (California Institute of Technology), Research Associate in Biology, *Department of Biology, California Institute of Technology, Pasadena, Calif.*

HOWELL, CHARLES DEWITT, A.B. (Oberlin), Ph.D. (Johns Hopkins), Associate Professor and Head of the *Department of Biology, Catawba College, Salisbury, N. C.*

HSIAO, SIDNEY CHIH TI, B.A. (Shanghai), M.S. (Yenching), Ph.D. (Harvard), *Department of Biology, Hua Chung College, Hsichow Uil Tali, Yunnan, China.*

HUDSON, GEORGE ELFORD, B.S. (Clemson), M.S., Ph.D. (Nebraska), Associate Professor of Zoology and Curator, Charles R. Conner Museum, *Department of Zoology, State College of Washington, Pullman, Wash.*

HUGGINS, RUSSELL ARNO, B.S. (Aurora College), Ph.D. (Western Reserve), Instructor in Biology, *Department of Biology, Eastern State Teachers College, Madison, S. D.*

HUMES, ARTHUR GROVER, B.A. (Brown), M.S. (Louisiana), Ph.D. (Illinois), Instructor in Zoology, University of Buffalo. (On leave for military service, '43, Lt. (j.g.), U. S. N. R., *Navy 3505 c/o Fleet Post Office, San Francisco, Calif.*)

HUNTER, WANDA SANBORN, B.A. (Wisconsin), M.S. (Illinois), Ph.D. (California, Los Angeles), Assistant Professor of Zoology, *Department of Zoology, University of Arizona, Tucson, Ariz.*

INGLE, LESTER, B.S. (Kansas State Teachers College), M.S., Ph.D. (Brown), Instructor in Biology, *Department of Biology, Fresno State College, Fresno, Calif.*

JOHNSON, J. GARTH, B.A. (Drake), M.S., Ph.D. (Iowa), Director of Microbiology, *Ortho Research Foundation, Linden, N. J.*

JOHNSON, LELAND PARRISH, B.S. (Monmouth College), M.S., Ph.D. (Iowa State), Associate Professor of Biology, *Department of Biology, Drake University, Des Moines, Iowa.*

JOHNSON, MAYNARD STICKNEY, A.B. (Bates), A.M., Ph.D. (Illinois), Associate Wildlife Technician, National Park Service, U. S. Department of Interior, *39 Summer St., Saugus, Mass.*

JOHNSON, MYCHYLE WELLINGTON, A.B. (Indiana Central), Ph.D. (Indiana), Assistant Professor of Zoology, *Department of Zoology, Duke University, Durham, N. C.*

JONES, CLAIRBORNE STRIBLING, B.A. (Hampden-Sydney), M.A., Ph.D. (Virginia), Assistant Professor of Zoology, *Department of Zoology, University of North Carolina, Chapel Hill, N. C.*

JONES, EDGAR PERCIVAL, B.S., M.S., Ph.D. (Pittsburgh), Associate Professor of Biology, Department of Biology, *University of Akron, Akron, Ohio.*

JONES, ROY WINFIELD, A.B. (Oklahoma City University), M.S. (Kansas State), Ph.D. (Oklahoma), Professor of Biology and Dean of the College, *Central State College, Edmond, Okla.*

KALISS, NATHAN, B.S. (College of the City of New York), M.A., Ph.D. (Columbia), Instructor in Biology, *Department of Biology, Brooklyn College, 1617 Ave. N., Brooklyn 30, N. Y.*

KAPLAN, HAROLD M., A.B. (Dartmouth), A.M., Ph.D. (Harvard), Professor of Physiology, *Department of Physiology, Middlesex University, Waltham, Mass.*

KAVANAGH, VIRGENE WARBRITTON, A.B., A.M., Ph.D., B.S. in Ed. (Missouri), Assistant, *New York Botanical Garden, Bronx, N. Y.*

KAYLOR, CORNELIUS TIMPSON, A.B. (Wesleyan), M.S. (Rutgers), Ph.D. (Princeton), Assistant Professor of Anatomy, *Medical College, Syracuse University, Syracuse, N. Y.*

KECK, WARREN N., B.A., M.S., Ph.D. (Iowa), Associate Professor of Biology, *Department of Biology, Coe College, Cedar Rapids, Iowa.*

KELSALL, MARGARET ASTON, B.A., M.A., Ph.D. (Colorado), Research Associate, *Hamilton Station, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.*

KITCHIN, IRWIN CLARK, B.S. (Wake Forest), Ph.D. (Freiburg), Associate Professor of Zoology, *Department of Zoology, University of North Carolina, Chapel Hill, N. C.*

KRAATZ, WALTER CHARLES, B.A. (Wisconsin), M.A., Ph.D. (Ohio State), Professor of Biology, *University of Akron, Akron, Ohio.*

LAIDLAW, HARRY H., JR., B.S., M.S. (Louisiana), Ph.D. (Wisconsin), *State Apiarist, Montgomery, Ala.* (On leave for military service, '43, *2nd Lt. Medical Administrative Corps, U. S. A. Station Hospital, S. A. A. F., Stuttgart, Ark.*)

LAMAR, JULE KITCHELL, B.S. (Birmingham-Southern College), Ph.D. (Chicago), Assistant Professor in Obstetrics and Gynecology, *The University of Texas, Medical Branch, Galveston, Texas.*

LANE, CHARLES EDWARD, B.A., M.A., Ph.D. (Wisconsin), Assistant Professor of Zoology, *University of Wichita, Wichita, Kan.*

LANSING, ALBERT I., A.B. (Pennsylvania), Ph.D. (Indiana), Assistant in Anatomy, *Department of Anatomy, Washington University School of Medicine, St. Louis, Mo.*

LE FEVRE, PAUL GREEN, A.B. (Johns Hopkins), Ph.D. (Pennsylvania), Instructor in Physiology, *College of Medicine, University of Vermont, 403 Colchester Ave., Burlington, Vt.*

Leonard, Arthur Byron, B.A. (Central State Teachers), M.A., Ph.D. (Kansas), Assistant Professor of Anatomy and Zoology, *Department of Zoology, University of Kansas, Lawrence, Kan.*

Leslie, Lawrence Dale, B.A., M.A. (Oregon), Ph.D. (Stanford), Associate Professor of Zoology, *San Jose State College, San Jose, Calif.*

Levenstein, Irving Max, A.B., M.S., Ph.D. (New York), *Leberco Laboratories, Bloomfield, N. J.*

Lewis, Lillian L. Burwell, B.S. (Howard), M.S., Ph.D. (Chicago), Professor of Zoology and Chairman of the Department, *Department of Zoology, Tillotson College, Austin 22, Texas.*

Libby, Raymond Loring, B.S., M.S., Ph.D. (Rutgers), Biophysicist, *American Cyanamid Company, Stamford, Conn.*

Light, V. Earl, A.B., M.S. (Lebanon Valley), Ph.D. (Johns Hopkins), Associate Professor of Biology, *Lebanon Valley College, R. No. 1, Annville, Pa.*

Lipsett, James Conley, B.S. (Lafayette), Ph.D. (Yale), Assistant Professor of Anatomy, *Department of Anatomy, University of Maryland, Baltimore. Md.*

Littleford, Robert Anthony, B.S., M.S., Ph.D. (Maryland), Assistant Professor of Zoology, *Department of Zoology, University of Maryland, College Park, Md.*

Lochhead, John Hutchison, M.A. (St. Andrews, Scotland), B.A., Ph.D. (Cambridge), Instructor in Zoology, *Department of Zoology, University of Vermont, Burlington, Vt.*

Lovell, Harvey Bulfinch, A.B. (Bowdoin), A.M., Ph.D. (Harvard), Associate Professor of Zoology, *Department of Biology, University of Louisville, Louisville, Ky.*

Lowrance, Edward Walton, A.B., A.M. (Utah), Ph.D. (Stanford), Associate Professor of Anatomy, *Department of Anatomy, University of Kansas, Lawrence, Kan.*

Lowrie, Donald C., B.S., Ph.D. (Chicago), Director, Museum of Natural History *of Academy of Science of Saint Louis, 4642 Lindell Blvd., Saint Louis, Mo.*

Lyman, Rufus Ashley, Jr., B.Sc., M.A. (Nebraska), Ph.D. (Johns Hopkins), Interne Naval Hospital, S. D. (j.g.) *3995 Crown Point Drive, San Diego 9, Calif.*

MacCardle, Ross C(layton), B.S. (Michigan), Ph.D. (Brown), Instructor and Research Associate, Department of Anatomy, *Washington University School of Medicine, St. Louis, Mo.* (On leave for military service, '42. Capt. A.C., U. S. A. Air Force, Aero Medical Laboratory, Wright Field, Dayton, Ohio.

Magalhaes, Hulda, B.S. (New Jersey College for Women), M.A. (Mount Holyoke), Ph.D. (Duke), Instructor in Zoology, *Department of Zoology, Duke University, Durham, N. C.*

Magruder, Samuel Rossington, B.S. (Kentucky), M.A., Ph.D. (Cincinnati), Assistant Professor of Anatomy, *Department of Anatomy, Tufts College Medical School, Boston, Mass.*

Maldonado-Koerdell, Manuel, B.S. (Mexico), Latin-American Guggenheim Fellow, *University of Kansas Museum of Natural History, Lawrence, Kan.*

Marvin, Horace Newell, B.A. (Morningside College), M.A., Ph.D. (Wisconsin), Instructor in Anatomy, *Department of Anatomy, School of Medicine, University of Arkansas, Little Rock, Ark.*

Meglitsch, Paul Allen, B.S., M.S., Ph.D. (Illinois), Instructor in Zoology, *Wright Junior College, Chicago, Ill.*

Merwin, Ruth M., A.B., A.M. (Mt. Holyoke), Ph.D. (Chicago), Instructor in Zoology, *Department of Zoology, Connecticut College, New London, Conn.*

Meyer, Marvin Clinton, B.S. (Missouri State College), M.A. (Ohio State), Ph.D. (Illinois), Professor of Zoology and Acting Chairman of the Department, New Jersey College for Women. (On leave for military service, '43, Capt. U. S. A., Sanitary Corps, *APO, 705 c/o Postmaster, San Francisco, Calif.*)

Michalski, Joseph Vitold, A.B. (Johns Hopkins), M.A., Ph.D. (Princeton), Aviation Physiologist, Army Air Corps, *Altitude Training Unit, Sq.t., Chathan Field, Ga.*

Miller, E. DeWitt, A.B. (Bridgewater), M.A., Ph.D. (Virginia), Associate Professor of Biology, *Madison College, Box 388, Harrisonburg, Va.*

Miller, Edwin Lynn, B.S. (Iowa Wesleyan), M.S., Ph.D. (Illinois), Associate Professor of Biology, *Department of Biology, Lawrence College, Appleton, Wis.*

Miller, Elwood Morton, B.S. (Bethany), M.S., Ph.D. (Chicago), Associate Professor of Zoology, *Department of Zoology, University of Miami, Coral Gables, Fla.*

Miller, Richard Avery, A.B. (Pittsburgh), M.S., Ph.D. (Iowa), Cytologist, *Station for Experimental Evolution, Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y.*

Mohr, John Luther, A.B. (Bucknell), Ph.D. (California), Research Associate in Zoology, Department of Zoology, University of California, Berkeley, and Research Associate in Tropical Diseases Survey, *Hoover Research Institute, Stanford University, Calif.*

Moment, Gairdner Bostwick, A.B. (Princeton), Ph.D. (Yale), Professor of Biology, *Department of Biology, Goucher College, Baltimore, Md.*

Moog, Florence, A.B. (New York), A.M., Ph.D. (Columbia), Instructor and Research Associate in Zoology, *Department of Zoology, Washington University, St. Louis 5, Mo.*

Moreland, George Edward, A.B. (Greenville), M.S. (Michigan), Ph.D. (Cornell), Professor of Biology, Chairman, Division of Science and Mathematics and Director of the Summer Session, *Houghton College, Houghton, N. Y.*

Morgan, Charles Frederick, A.B. (Wichita), Ph.D. (Chicago), Assistant Professor of Pharmacology and Materia Medica, *Department of Pharmacology, Georgetown University School of Medicine, Washington, D. C.*

Moritz, Charles Edgar, A.B. (Dartmouth), Ph.D. (California), Marine Biologist, *Naval Ordnance Laboratory, 1664 Steele St., Denver 6, Colo.*

Myers, Hugh I(rvin), B.A. (Mississippi), M.A. (Richmond), Ph.D. (Brown), *School of Dentistry, University of Kansas City, Kansas City, Mo.*

Myers, Raymond J., A.B. (Heidelberg), Ph.D. (Pennsylvania), Assistant Professor of Zoology, *Department of Zoology, Colgate University, Hamilton, N. Y.*

NAHM, LAURA JULIA, B.S. in Ed., A.B., A.M., Ph.D. (Missouri), Assistant Professor of Biology, *Central Missouri State Teachers College, Warrensburg, Mo.*

NEAVE, FERRIS, B.Sc., M.Sc. (Manchester), Assistant Biologist, Fisheries Research Board of Canada, *Pacific Biological Station, Nanaimo, British Columbia, Canada.*

NESTER, HENRY GEORGE, A.B. (Butler), A.M., Ph.D. (Indiana), Professor and Head of the *Department of Physiology and Health, Butler University, Indianapolis, Ind.*

NICKERSON, MARK, A.B. (Linfield), Sc.M. (Brown), Ph.D. (Johns Hopkins), Instructor in Pharmacology, *Department of Pharmacology, University of Utah School of Medicine, Salt Lake City, Utah.*

NOBLE, ELMER R., A.B., M.A., Ph.D. (California), Associate Professor of Biology, *Santa Barbara College, University of California, Santa Barbara, Calif.*

NOBLE, GLENN ARTHUR, A.B., M.A. (California), Ph.D. (Stanford), Department of Zoology, *San Francisco Junior College, Ocean and Phelan Ave., San Francisco, Calif.*

NORRIS, CHARLES H., B. S. (Hamilton), Ph.D. (Princeton), Assistant Professor of Biology, *Department of Biology, University of Colorado, Boulder, Colo.*

NUNNEMACHER, RUDOLPH FINK, B.S. (Kenyon), M.A., Ph.D. (Harvard), Associate Professor of Biology, *Clark University, Worcester, Mass.*

O'BRIEN, JOHN PATRICK, B.S. (Illinois), Ph.D. (Johns Hopkins), Professor of Biology, *Gannon College, Erie, Pa.*

ODIORNE, JOSEPH MILTON, B.S. (Bowdoin), A.M., Ph.D. (Harvard), Assistant Professor of Anatomy, *Department of Anatomy, Georgetown University School of Medicine, Washington, D. C.*

OLIPHANT, JOSEPH F(REDERICK), A.B. (Wabash), Ph.D. (Johns Hopkins), Assistant Professor of Biology, *Stanford University, Stanford University, Calif.*

OLSON, MAGNUS, B.A. (St. Olaf), M.A., Ph.D. (Minnesota), Instructor in Zoology, *Department of Zoology, University of Minnesota, Minneapolis, Minn.*

OSTERUD, HJALMAR LAURITZ, A.B., A.M. (Washington), Ph.D. (Minnesota), Professor of Anatomy and Head of the Department, *Medical College of Virginia, Richmond, Va.*

OTIS, ARTHUR BROOKS, A.B. (Maine), M.Ed. (Springfield), Sc.M., Ph.D. (Brown), Instructor in Physiology, *Department of Physiology, The University of Rochester, School of Medicine, Rochester, N. Y.*

OWEN, BRADFORD BRECKENRIDGE, B.A., M.A. (Williams College), M.A., Ph.D. (Harvard), Instructor in Biology, *Long Island University, Brooklyn, N. Y.*

PEARSON, JAY FREDERICK WESLEY, B.S., M.S. (Pittsburgh), Ph.D. (Chicago), Professor of Zoology and Dean of Administration, University of Miami, *321 S.W. 32 Rd., Miami, Fla.* (On leave for military service, '42. Capt., U. S. A. Air Force.)

PEASE, DANIEL CHAPIN, A.B. (Yale), M.S. (California Institute of Technology), Ph.D. (Princeton), *University of Southern California, School of Medicine, Los Angeles, Calif.*

PERROT, MAX, Ph.D. (Geneva), Instructor in Zoology, *Department of Zoology, University of Missouri, Columbia, Mo.*

PIERCE, MADELENE EVANS, A.B., A.M., Ph.D. (Radcliffe), Associate Professor of Zoology, *Vassar College, Poughkeepsie, N. Y.*

Pitelka, Frank A(lois), B.S. (Illinois), Ph.D. (California), Lecturer in Zoology, Department of Zoology, and Assistant Curator of Birds, *Museum of Vertebrate Zoology, University of California, Berkeley 4, Calif.*

Pollister, Priscilla F., A.B. (Bates), A.M., Ph.D. (Columbia), Assistant Professor of Biology, *Department of Biology, Brooklyn College, Bedford Ave. and Ave. H, Brooklyn, N. Y.*

Pomerat, Gerard Roland, A.B. (Clark), A.M., Ph.D. (Harvard), Instructor in Biology, Biological Laboratories, *Harvard University, Cambridge, Mass.*

Porter, Keith Roberts, B.Sc. (Acadia), A.M., Ph.D. (Harvard), Research Assistant, *Rockefeller Institute, New York, N. Y.*

Powers, Edward Lawrence, Jr., B.S. (Charleston), Ph.D. (Johns Hopkins), *151 Moultrie St., Charleston 19, S. C.*

Quiring, Daniel Paul, A.B. (Oberlin), M.A. (Illinois), Ph.D. (Western Reserve), Associate Professor of Biology, Western Reserve University, and Head of Anatomy Division, Cleveland Clinic Foundation, *Western Reserve University, Cleveland, Ohio.*

Ray, Ovid Malcolm, B.A., M.A. (Drake), Ph.D. (Iowa), Assistant Professor of Physiology and Zoology, *North Dakota Agricultural College, Fargo, N. D.*

Renshaw, Birdsey, S.B., M.A., Ph.D. (Harvard), Assistant Professor, *Wright Laboratory, Oberlin College, Oberlin, Ohio.*

Rice, Nolan Ernest, A.B. (Kentucky), A.M., Ph.D. (Duke), *Head of Culture Department, Carolina Biological Supply Company, Elon College, N. C.*

Ring, John Robert, A.B. (Illinois), Sc.M., Ph.D. (Brown), Instructor in Microanatomy, *Department of Microanatomy, Saint Louis University School of Medicine, 1402 South Grand Blvd., St. Louis 4, Mo.*

Ris, Hans, Ph.D. (Columbia), Assistant in Physiology, *Rockefeller Institute, 66th Street and York Avenue, New York, N. Y.*

Robeson, John Maxwell, B.S., M.S., Ph.D. (Virginia), Associate Professor of Zoology, *Department of Zoology, Syracuse University, Syracuse, N. Y.*

Robinson, True William, B.S. (California Institute of Technology), M.A., Ph.D. (Harvard), Associate in Zoology, *345 Natural History Building, University of Illinois, Urbana, Ill.* (On leave for military service, '43, *2nd Lt. U. S. A. Air Force, Aviation Physiologist, Altitude Training Section, Muroc AAF, Calif.*)

Rodgers, Thomas Latham, B.A. (San Jose), M.A. (California), Assistant Professor of Biology, *Department of Biology, Chico State College, Chico, Calif.*

Roeder, Kenneth David, B.A., M.A. (Cambridge, England), Assistant Professor of Biology, *Tufts College, Medford, Mass.*

Rogers, Philip V., A.B., A.M. (Hamilton), Ph.D. (Yale), Assistant Professor of Biology, *Hamilton College, Clinton, N. Y.*

Roosen-Runge, Edward C., Dr.med. (Hamburg), *Photographic Laboratory, Brown University, Providence, R. I.*

Root, Clinton William, A.B., A.M. (Oberlin), Ph.D. (Princeton), Assistant Professor of Zoology, *Lyman Hall, Syracuse University, Syracuse, N. Y.*

Rostorfer, Howard Hayes, B.A. (Wittenberg), M.A., Ph.D. (Ohio State), Assistant Professor of Physiology, *Department of Physiology and Pharmacology, University of Arkansas School of Medicine, Little Rock, Ark.*

ROTHCHILD, IRVING, B.A., M.A., Ph.D. (Wisconsin), Assistant Physiologist, U. S. Department of Agriculture, *36 C Crescent Road, Greenbelt, Md.*

RUBIN, DAVID, B.S., Ph.D. (Chicago), Medical School, Army Specialized Training, *4609 N. Spaulding Ave., Chicago 25, Ill.*

RUSSELL, ELIZABETH BUCKLEY SHULL, A.B. (Michigan), M.A. (Columbia), Ph.D. (Chicago), Independent Investigator, *Jackson Laboratory, Bar Harbor, Me.*

RYERSON, DWIGHT LEONARD, A.B., M.S. (Arizona), Ph.D. (California, Los Angeles), Instructor in Anatomy, *Department of Anatomy, University of Arkansas, School of Medicine, Little Rock, Ark.*

SAWIN, HORACE JOHN, A.B. (Earlham College), A.M., Ph.D. (Harvard), Research Consultant in Paleontology, *State Museum, 1904 San Gabriel Ave., Austin, Texas.*

SAWYER, ELIZABETH LOUISE, A.B. (Maine), A.M. (Michigan), Ph.D. (Missouri), Associate Professor of Biology, *Department of Biology, Rockford College, Rockford, Ill.*

SCHELL, MARGARET WOOD, A.B., M.A., Ph.D. (California), Assistant Professor of Zoology, *University of Utah, Salt Lake City, Utah.*

SCHOENBORN, HENRY WILLIAM, A.B. (DePauw), Ph.D. (New York), Assistant Professor of Zoology, *Division of Biological Science, University of Georgia, Athens, Ga.*

SCOTT, SISTER FLORENCE MARIE, A.B. (Seton Hill), M.A., Ph.D. (Columbia), Professor of Biology, *Seton Hill College, Greensburg, Pa.*

SCOTT, WALTER JAMES, Ph.B. (Lafayette), Ph.D. (Pennsylvania), Associate Professor of Zoology, *Swarthmore College, Swarthmore, Pa.*

SHAFTESBURY, ARCHIE D., A.B. (Southwestern), Ph.D. (Johns Hopkins), Associate Professor of Zoology, *Woman's College of the University of North Carolina, Greensboro, N. C.*

SHEEHAN, JOHN FRANCIS, B.S., M.S. (New Hampshire), Ph.D. (Iowa), Assistant Professor of Biology, *Department of Biology, Creighton University, Omaha 2, Nebr.*

SHALER, SIMON, A.B., M.A., Ph.D. (Columbia), Laboratory of Biophysics, *Columbia University, New York, N. Y.*

SICHEL, ELSA KEIL, B.S. (Elmira), M.A. (Brown), Professor of Biology and Head of the Biology Department, *Trinity College, Burlington, Vt.*

SIMMONS, GEORGE FINLAY, B.A., M.A. (Texas), Ph.D. (Chicago), Assistant Professor of Anatomy, *Loyola University School of Medicine, 706 S. Wolcott Ave., Chicago 12, Ill.*

SMITH, CHARLES CLINTON, B.S. (Central State Teachers College, Oklahoma), M.S., Ph.D. (Oklahoma), District Conservationist, U. S. Soil Conservation Service, *c/o Soil Conservation Service, Alice, Texas.*

SMITH, FREDERICK GEORGE WALTON, B.Sc., Ph.D. (London), A.R.C.S. (Royal College of Science), Associate Professor of Zoology, Director Marine Laboratory, University of Miami, *Coral Gables, Fla.*

SOLINGER, JULIAN L., Ph.B., Sc.M., Ph.D. (Brown), B.Ed. (Rhode Island College of Education), Assistant Professor of Biology, *Simmons College, 300 The Fenway, Boston, Mass.*

SONNENBLICK, BENJAMIN P., B.S. (College of the City of New York), M.S., Ph.D. (New York), Associate in Biology, *Department of Biology, Queens College, Flushing, N. Y.*

SPIETH, HERMAN T., A.B. (Indiana Central), Ph.D. (Indiana), Assistant Professor of Zoology, *Biology Department, College of the City of New York, 139th St. and Convent Ave., New York, N. Y.* (On leave for military service, '44, Lt. A. C., *Navigation Department, Cochran Field, Macon, Ga.*)

SPOOR, WILLIAM ARTHUR, B.S. (Washington), Ph.D. (Wisconsin), Assistant Professor of Zoology, *Department of Zoology, University of Cincinnati, Cincinnati, Ohio.*

SPOTKOV, ELIAS MORLENN, B.S. (College of the City of New York), M.S., Ph.D. (New York), *U. S. Office of Censorship, 120 E. Myrtle St., San Antonio, Texas.*

STEFFENS, HERMAN WALTER, B.S., M.S. (Idaho), Ph.D. (Harvard), Associate Professor of Zoology, and Acting Head of Department of Zoology, *University of Idaho, Moscow, Idaho.*

STREET, SIBYL FAWCETT (MRS. ROBERT RAMSEY), A.B. (Vassar), Ph.D. (Chicago), *c/o Robert Ramsey, Department of Physiology, Medical College of Virginia, Richmond, Va.*

STULTZ, WALTER ALVA, B.A. (Acadia), Ph.D. (Yale), Associate Professor of Anatomy, *College of Medicine, University of Vermont, Burlington, Vt.*

SWEET, HELEN E., B.A. (Pomona), M.A. (Claremont), Ph.D. (Chicago), Associate Professor of Biology, *Santa Barbara College, University of California, Santa Barbara, Calif.*

TAHMISIAN, THEODORE NEWTON, B.A. (Fresno State College), M.S., Ph.D. (Iowa), Aviation Physiologist, U. S. A., Ist Lt., A.C. *Altitude Training Unit, Drew Field, Tampa, Fla.*

TARTAR, VANCE, B.S., M.S. (Washington), Ph.D. (Yale), *Biologist, State Oyster Laboratory, Gig Harbor, Wash.*

TASKER, ROY C., A.B. (Hillsdale), A.M. (Michigan), Ph.D. (Cornell), Associate Professor of Zoology, *Bucknell University, Lewisburg, Pa.*

TAYLOR, ALFRED, B.A. (Oregon), M.A., Ph.D. (Oregon State College), *Biochemical Institute, University of Texas, Austin, Texas.*

TEST, FREDERICK H(AROLD), B.S. (Purdue), A.M. (Cornell), Ph.D. (California), Assistant Professor of Zoology, *Department of Zoology, University of Michigan, Ann Arbor, Mich.*

TE WINKEL, LOIS EVELYN, A.B. (Oberlin), A.M., Ph.D. (Columbia), Associate Professor of Zoology, *Department of Zoology, Smith College, Northampton, Mass.*

THOMAS, THURLO BATES, S.B. (Wooster), M.A. (Oberlin), Ph.D. (Harvard), Instructor in Anatomy, *Department of Anatomy, University of Texas, Medical Branch, Galveston, Texas.*

THORNTON, CHARLES STEAD, A.B. (Harvard), A.M., Ph.D. (Princeton), Associate Professor of Biology and Chairman of the Department, *Kenyon College, Gambier, Ohio.*

TIPTON, SAMUEL RIDLEY, A.B. (Mercer), Ph.D. (Duke), Instructor in Physiology, *Department of Physiology, Medical College of Alabama, Birmingham 5, Ala.*

TODD, ARLIE C., B.A. (Kalamazoo), M.S. (Kansas State), Ph.D. (Nebraska), Instructor in Zoology, *Department of Zoology, Agricultural Experiment Station, University of Tennessee, Knoxville, Tenn.*

TODD, ROBERT EMERSON, JR., B.S. (Bowdoin), M.A., Ph.D. (Harvard), Assistant Professor in Zoology, Colgate University, *Box 195, Hamilton, N. Y.*

TRUITT, R. V. T., B.S., M.S. (Maryland), Ph.D. (American), Director, Maryland Department of Research and Education; Director, Chesapeake Biological Laboratory, *Solomons Island, Md.*

UHRICH, JACOB, A.B. (Doane), M.A. (Nebraska), Ph.D. (Chicago), Professor of Biology and Chairman of the Department, *Trinity University, San Antonio, Texas.*

VENARD, CARL ERNEST, B.A., M.S. (Ohio), Ph.D. (New York), Assistant Professor of Zoology and Entomology, *Department of Zoology and Entomology, Ohio State University, Columbus 10, Ohio.*

VOGEL, HOWARD H., JR., A.B. (Bowdoin), M.A., Ph.D. (Harvard), Associate Professor of Zoology and Acting Chairman of the Department, *Wabash College, Crawfordsville, Ind.*

WAGNER, ROBERT PHILIP, B.S. (College of the City of New York), Ph.D. (Texas), Assistant Professor of Zoology, *Department of Zoology and Physiology, University of Texas, Austin, Texas.*

WALKER, PAUL ANDREW, A.B. (Bowdoin), Ph.D. (Harvard), Professor of Biology and Chairman of the Department, *Randolph-Macon Woman's College, Lynchburg, Va.*

WALKER, ROLAND, A.B., M.A. (Oberlin), Ph.D. (Yale), Assistant Professor of Biology, *Department of Biology, Rensselaer Polytechnic Institute, Troy, N. Y.*

WATERMAN, TALBOT HOWE, A.B., M.A., Ph.D. (Harvard), Secretary, Committee on Research, *c/o Osborn Zoological Laboratory, Yale University, New Haven, Conn.*

WAUGH, DAVID FLOYD, A.B., Ph.D. (Washington), Assistant Professor of Physical Biology, *Department of Biology and Biological Engineering, Massachusetts Institute of Technology, Cambridge, Mass.*

WEATHERBY, JESSE HOWELL, B.S. (Mississippi A. and M.), M.A., Ph.D. (Johns Hopkins), Research Associate, *Department of Pharmacology, Medical College of Virginia, Richmond, Va.*

WELTY, J(OEL) CARL, A.B. (Earlham), A.M. (Haverford), Ph.D. (Chicago), Professor of Zoology and Head of the Department of Biology, Beloit College, *Route 1, Beloit, Wis.*

WHEELER, NORMAN C., A.B. (Berea), M.S., Ph.D. (Purdue), 1st Lt. Sn. C.A.U.S., *Medical Nutrition Laboratory, 1849 W. Pershing Rd., Chicago 9, Ill.*

WHITTINGHILL, MAURICE, A.B. (Dartmouth), Ph.D. (Michigan), Associate Professor of Zoology, *Department of Zoology, University of North Carolina, Chapel Hill, N. C.*

WILDER, MAGEL CRAIG, A.B., A.M. (Brown), Ph.D. (Cornell), Assistant Professor of Biology, Pembroke College, Brown University, *Olneyville Station, R.F.D. No. 2, Providence, R. I.*

WILHELMI, RAYMOND WILLIAM, A.B. (Nebraska Wesleyan), Ph.D. (New York), Assistant Professor of Zoology, *Department of Zoology, University of Missouri, Columbia, Mo.*

WILSON, JOHN WOODROW, B.S., Ph.D. (Duke), Instructor in Zoology, *Department of Zoology, Duke University, Durham, N. C.*

WILSON, LOUISE PALMER, A.B. (Southwestern), M.S., Ph.D. (Pennsylvania), Associate Professor of Zoology, *Department of Zoology and Physiology, Wellesley College, Wellesley, Mass.*

WIMSATT, WILLIAM ABELL, A.B., Ph.D. (Cornell), Assistant Professor of Zoology, *Stimson Hall, Cornell University, Ithaca, N. Y.*

WINSOR, CHARLES P., A.B., B.S., Ph.D. (Harvard), Assistant Professor of Biostatistics, *School of Hygiene and Public Health, The Johns Hopkins University, Baltimore 5, Md.*

WOLF, E(MIL) ALFRED, Ph.D. (Breslau), Associate Professor of Biology, *Department of Biology, University of Pittsburgh, Pittsburgh, Pa.*

WOLF, LEONARD NICHOLAS, B.S. (Saint Vincent College), M.S., Ph.D. (Pittsburgh), Professor of Biology, *University of Scranton, Scranton 3, Pa.*

WOLF, OPAL MARIE, A.B. (Southwestern), A.M., Ph.D. (Wisconsin), Assistant Professor of Physiology, *Woman's College of the University of North Carolina, Greensboro, N. C.*

WOLFSON, ALBERT, B.S. (Cornell), Ph.D. (California), Instructor in Zoology, *Department of Zoology, Northwestern University, Evanston, Ill.*

WOODARD, THOMAS MARION, A.B., A.M. (Vanderbilt), Ph.D. (Princeton), Associate Professor of Biology, *Vanderbilt University, Nashville, Tenn.*

WOLTERINK, LESTER FLOYD, A.B. (Hope College), M.A., Ph.D. (Minnesota), Instructor in Physiology, *Department of Physiology and Pharmacology, Michigan State College, East Lansing, Mich.*

WRIGHT, PHILIP LINCOLN, B.S., M.S. (New Hampshire), Ph.D. (Wisconsin), Assistant Professor of Zoology, *Department of Zoology, Montana State University, Missoula, Mont.*

WULFF, VERNER JOHN, A.B. (Wayne), M.A. (Northwestern), Ph.D. (Iowa), Research Assistant in Zoology, *Department of Zoology, State University of Iowa, Iowa City, Iowa.* (On leave for military service, '42. Lt., U. S. A. Air Force.)

YANCEY, PATRICK H., A.B., A.M. (Gonzaga), Ph.D. (St. Louis), Professor of Biology, *Spring Hill College, Mobile, Ala.*

YEAKEL, ELEANOR HUGINS, A.B., M.A., Ph.D. (Bryn Mawr), Fellow in Biology, *The Wistar Institute of Anatomy and Biology, Philadelphia 4, Pa.*

YEATTER, R(ALPH) E(MERSON), B.S. (Michigan State), M.S., Ph.D. (Michigan), Game Specialist, Illinois Natural History Survey, *226 Natural Resources Building, Urbana, Ill.*

YOUNG, DIXIE, A.B., A.M. (Oklahoma), Ph.D. (Yale), Associate Professor of Zoology, *University of Oklahoma, Norman, Okla.*

ZALESKY, MOSES, A.B., Ph.D. (Chicago), Director, Bureau of Jewish Education, Cincinnati, Ohio, *658 Rockdale Ave., Cincinnati 29, Ohio.*

ZWEIFACH, BENJAMIN WILLIAM, B.S. (College of the City of New York), M.S., Ph.D. (New York), Research Associate in Biology, New York University, *26 Washington Place, New York, N. Y.*

ZWILLING, EDGAR, B.S. (Brooklyn College), M.A., Ph.D. (Columbia), Instructor, Storrs Agricultural Experiment Station, *University of Connecticut, Storrs, Conn.*

Totals

Life members	2
Active members	775
Associate members	303
Grand total	1080

INDEX

ACTIVITY and action currents of heart and skeletal muscle in tissue culture, comparison of 125
Adrenal cortex, embryonic and postnatal development of the 391
Alkaline phosphatase activity following various methods of histologic fixation, intracellular distribution of 159
American Association of Anatomists. Proceedings of the fifty-ninth session, officers and list of members 201
American Society of Zoologists...... Officers, constitution, by-laws, proceedings and list of members. Forty-second annual meeting, Saint Louis 461
Amoeboid movement of the mammalian leukocyte in tissue culture 177
"Anatomical position," is concept of, still tenable? 81
Anatomical specimens, method of duplicating 119
Anatomy and function of the foreleg in salamander locomotion 257
Anomalies in the small intestine and cecum of inbred strains of mice . 1
Anomalous middle meningeal artery .. 347
Anomaly, arterial, in the fetal pig ... 53
Anterior hypophysis of the dog, cytological study of the 11
Appendix, arterial supply of the vermiform 457
Arterial supply of the vermiform appendix 457
Artery, anomalous middle meningeal . 347
Atkinson, William B. and James H. Leathem. Day to day level of estrogen and progestin during lactation in the mouse 147

BENSLEY, R. R. *Book Review.* Physical chemistry of cells and tissues by Rudolf Höber and others . 75
Bevelander, G. See Johnson, P. L. . 193
Bishop, D. H. and J. H. Leathem. Response of prepuberal male mice to equine gonadotropin 313
Bone marrow of man. The conical openings in the wall of venous sinusoids and their relation to erythrogenic capillaries in the 379
Book Reviews 73

CAT. Postpartum history of the corpus luteum of the 29
Cecum and small intestine, anomalies in the, of inbred strains of mice 1
Cell, fourth, type in the anterior hypophysis of the dog . .. 11
Colloid, intracellular, of the thyroid gland, action of thiourea on 365
Conceptus during pregnancy, relation of hydrostatic conditions in the uterus to size and shape of the 283
Conical openings in the wall of venous sinusoids and their relation to the so-called erythrogenic capillaries in the bone marrow of man ... 379
Containers for human antomy, glass and steel 303
Corpus luteum of the cat, postpartum history of the 29

DAWSON, ALDEN B. The postpartum history of the corpus luteum of the cat 29
De Bruyn, P. P. H. Amoeboid movement of the mammalian leukocyte in tissue culture 177
Dentin, old and new concepts on the structure of 449
Differentiation, comparison of, of heart and skeletal muscle in tissue culture 125
Dog, cytological study of the anterior hypophysis of the, with reference to presence of a fourth cell type 11
Dorsal interosseous muscle of the hand, innervation of 7

EMBRYO, human, fusion of notochord to neural tube in a 307
Embryonic and postnatal development of adrenal cortex, particularly the zona glomerulosa and accessory nodules 391
Emmel, Victor M. Intracellular distribution of alkaline phosphatase activity following various methods of histologic fixation 159
Emory, Louis. Method of duplicating anatomical specimens . 119
Endochondral ossification, effect of male sex hormone on 97
Epiphyseal union in the opossum, sequence of 353
Erythrogenic capillaries in the bone marrow of man, conical openings in the wall of venous sinusoids and their relation to . . 379
Essenberg, J. M. Glass and steel containers for human anatomy 303
Estrogen and progentin, day to day level of, during lactation in the mouse 147
Evans, F. Gaynor. Anatomy and function of the foreleg in salamander locomotion 257

FAIN, W. R. See Hartmann, J. Francis 11
Farris, Edmond J. The time of ovulation in the monkey 337

Fibers. Silver-gelatin method for staining 297
Fixation. Intracellular distribution of alkaline phosphatase activity following various methods of histologic 159
Foreleg in salamander locomotion, anatomy and function of the 257
Form and behavior of adult mammalian skeletal muscle in vitro 321
Function and anatomy of the foreleg in salamander locomotion 257
Fundulus heteroclitus, a case of atypical twinning in 67
Fusion of notochord to neural tube in a human embryo of the sixth week 307

GALL bladder, a simian, deeply cleft, bilobed, with a phrygian cap 423
Glass and steel containers for human anatomy 303
Glycogen phosphatase in the developing hair 193
Goland, Philip P. See Lewis, Margaret R. 89
Gonadotropin, equine, response of prepuberal male mice to 313
Grasso, Rafael. Action of thiourea on the intracellular colloid of the thyroid gland 365
Gregg, Robert E. An arterial anomaly in the fetal pig 53
Growth, comparison of, of heart and skeletal muscle in tissue culture 125
Growth of sarcomata in mice, in vivo staining and retardation of 89
Gruenwald, Peter. Embryonic and postnatal development of the adrenal cortex, particularly the zona glomerulosa and accessory nodules 391

HAIR, glycogen phosphatase in the developing 193
Hand. Innervation of the first dorsal interosseous muscle of the 7
Hartmann, J. Francis, W. R. Fain and J. M. Wolfe. Cytological study of the anterior hypophysis of the dog with particular reference to the presence of a fourth cell type 11
Heart and skeletal muscle in tissue culture, comparison of growth, differentiation, activity and action currents of 125
Histologic fixation. Intracellular distribution of alkaline phosphatase activity following various methods of 159
Hormone, male sex, effect of, on endochondral ossification 97
Hydrostatic conditions in the uterus to size and shape of the conceptus during pregnancy, relation of 283
Hypophysis, anterior, of the dog, cytological study of the 11
Human anatomy, glass and steel containers for 303
Human gall bladder with a phrygian cap 423
Hurme, V. O. Old and new concepts on the structure of dentin 449

INNERVATION of the first dorsal interosseous muscle of the hand 7
Intestine and cecum of inbred strains of mice, anomalies in the small 1
Intracellular colloid of the thyroid gland, action of thiourea on the 365
Intracellular distribution of alkaline phosphatase activity following various methods of histologic fixation 159
In vivo staining and retardation of growth of sarcomata in mice 89

JOHNSON, P. L. and G. Bevelander. Glycogen phosphatase in the developing hair 193

KELSALL, Margaret A. Anomalies in the small intestine and cecum of inbred strains of mice 1
Kirkman, Hadley. A simian, deeply cleft, bilobed gall bladder with a "phrygian cap" 423

LACHMAN, Ernest. Is the concept of the "anatomical position" still tenable? A contribution to the subject of anatomical nomenclature 81
Lactation in the mouse, day to day level of estrogen and progestin during 147
Leathem, James H. See Atkinson, William B. 147
Leathem, J. H. See Bishop, D. H. 313
Leukocyte, mammalian, in tissue culture, amoeboid movement of the 177
Lewis, Margaret R., Henry A. Sloviter and Philip P. Goland. In vivo staining and retardation of growth of sarcomata in mice 89
Locomotion, anatomy and function of the foreleg in salamander 257
Low, Frank N. Anomalous middle meningeal artery 347

MALE sex hormone, effect of, on endochondral ossification 97
Mammalian leukocyte in tissue culture, amoeboid movement of the 177
Mammalian skeletal muscle in vitro, form and behavior of adult 321
Man. Conical openings in the walls of venous sinusoids and their relation to the erythrogenic capillaries in the bone marrow of 379
Method of duplicating anatomical specimens 119
Mice. Anomalies in the small intestine and cecum of inbred strains of 1
Mice. In vivo staining and retardation of growth of sarcomata in 89
Mice, prepuberal male, response of, to equine gonadotropin 313
Monkey, time of ovulation in the 337
Mouse, day to day level of estrogen and progestin during lactation in the 147
Murray, Margaret R. See Pogogeff, Irene A. 321
Muscle, mammalian skeletal, form and behavior of adult 321
Muscle of the hand, innervation of the first dorsal interosseous 7

NEURAL tube in a human embryo, fusion of notochord to 307
Notochord, fusion of, to neural tube in a human embryo 307
Nomenclature, anatomical, contribution to the subject 81

O'NEILL, Sarah Lea. See Pearson, Anthony A. 297
Opossum, sequence of epiphyseal union in the 353

Oppenhiemer, Jane M. Case of atypical twinning in Fundulus heteroclitus ... 67
Ossification, endochondral, effect of male sex hormone on ... 97
Ovulation, time of, in the monkey ... 337

PATTEN, Bradley M. Fusion of notochord to neural tube in a human embryo of the sixth week 307
Pearson, Anthony A. and Sarah Lea O'Neill. Silver-gelatin method for staining fibers ... 297
Phosphatase, glycogen, in the developing hair ... 193
Phrygian cap, a simian, deeply cleft, bilobed gall bladder with a ... 423
Pig, fetal, an arterial anomaly in the .. 53
Pogogeff, Irene A. and Margaret R. Murray. Form and behavior of adult mammalian skeletal muscle in vitro ... 321
Postpartum history of the corpus luteum of the cat ... 29
Pregnancy. Relation of hydrostatic conditions in the uterus to size and shape of the conceptus during 283
Prepuberal male mice, response of, to equine gonadotropin ... 313
Proceedings of the fifty-ninth session, officers and list of members of the American Association of Anatomists ... 201
Proceedings of the forty-second annual meeting of the American Society of Zoologists ... 461
Progestin and estrogen, day to day level of, during lactation in the mouse ... 147

RETARDATION of growth of sarcomata in mice ... 89
Reynolds, S. R. M. Relation of hydrostatic conditions in the uterus to size and shape of the conceptus during pregnancy. A concept of uterine accommodation ... 283

SALAMANDER locomotion, anatomy and function of the foreleg in 257
Sarcomata in mice, in vivo staining and retardation of growth of .. 89
Schleicher, Emil Maro. The conical openings in the wall of venous sinusoids and their relation to the so-called erythrogenic capillaries in the bone marrow of man . 379
Sequence of epiphyseal union in the opossum ... 353
Sex hormone, male, effect of, on endochondral ossification ... 97
Shaw, M. A., and Mubarika Shah. Arterial supply of the vermiform appendix ... 457
Silberberg, Martin and Ruth Silberberg. Further investigations on the effect of male sex hormone on endochondral ossification ... 97
Silver-gelatin method for staining fibers 297
Skeletal and heart muscle in tissue culture, comparison of growth, differentiation, activity and action current of ... 125
Skeletal muscle, mammalian, in vitro, form and behavior of adult 321
Sloviter, Henry A. See Lewis, Margaret R. ... 89
Staining fibers, silver-gelatin method for 297
Steel and glass containers for human anatomy ... 303
Structure of dentin, old and new concepts on the ... 449
Sunderland, Sydney. The innervation of the first dorsal interosseous muscle of the hand ... 7
Szepsenwol, J. Comparison of growth, differentiation, activity and action currents of heart and skeletal muscle in tissue culture ... 125

THIOUREA, action of, on the intracellular colloid of the thyroid gland 365
Thyroid gland, action of thiourea on the intracellular colloid of the . 365
Tissue culture, amoeboid movement of the mammalian leukocyte in ... 177
Twinning, atypical, in Fundulus heteroclitus ... 67

UTERUS, relation of hydrostatic conditions in the, to size and shape of the conceptus during pregnancy 283

VENOUS sinusoids, conical openings in the wall of, and their relation to the erythrogenic capillaries in the bone marrow of man . . 379
Vermiform appendix, arterial supply of 457

WALLIS, Wilson D. *Book Review.* Introduction to physical anthropology, by M. F. Ashley Montagu 77
Washburn, S. L. The sequence of epiphysial union in the opossum 353
Wells, L. J. *Book Review.* Biological actions of sex hormones, by Harold Burrows ... 73
Wolfe, J. M. See Hartmann, J. Francis ... 11

ZONA glomerulosa and accessory glands in the development of the adrenal cortex ... 391

41A85

COUNTWAY LIBRARY
HC 6DZR